ACS SYMPOSIUM SERIES 338

Solving Hazardous Waste Problems

Learning from Dioxins

Jurgen H. Exner, EDITOR
International Technology Corporation

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.fw001

Developed from a symposium sponsored
by the Division of Environmental Chemistry
at the 191st Meeting
of the American Chemical Society,
New York, New York,
April 13-18, 1986

American Chemical Society, Washington, DC 1987

Library of Congress Cataloging-in-Publication Data

Solving hazardous waste problems.
(ACS symposium series, ISSN 0097-6156; 338)

"Developed from a symposium sponsored by the Division of Environmental Chemistry at the 191st Meeting of the American Chemical Society, New York, New York, April 13-18, 1986."

Includes bibliographies and indexes.

1. Dioxins—Environmental aspects—Congresses. 2. Hazardous waste sites—Environmental aspects—Congresses. 3. Environmental chemistry—Congresses.

I. Exner, Jurgen H. II. American Chemical Society. Division of Environmental Chemistry. III. American Chemical Society. Meeting (191st: 1986: New York, N.Y.) IV. Series.

TD196.C5S64 1987 363.7'384 87-1389
ISBN 0-8412-1025-X

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.fw001

Copyright © 1987

American Chemical Society

All Rights Reserved. The appearance of the code at the bottom of the first page of each chapter in this volume indicates the copyright owner's consent that reprographic copies of the chapter may be made for personal or internal use or for the personal or internal use of specific clients. This consent is given on the condition, however, that the copier pay the stated per copy fee through the Copyright Clearance Center, Inc., 27 Congress Street, Salem, MA 01970, for copying beyond that permitted by Sections 107 or 108 of the U.S. Copyright Law. This consent does not extend to copying or transmission by any means—graphic or electronic—for any other purpose, such as for general distribution, for advertising or promotional purposes, for creating a new collective work, for resale, or for information storage and retrieval systems. The copying fee for each chapter is indicated in the code at the bottom of the first page of the chapter.

The citation of trade names and/or names of manufacturers in this publication is not to be construed as an endorsement or as approval by ACS of the commercial products or services referenced herein; nor should the mere reference herein to any drawing, specification, chemical process, or other data be regarded as a license or as a conveyance of any right or permission, to the holder, reader, or any other person or corporation, to manufacture, reproduce, use, or sell any patented invention or copyrighted work that may in any way be related thereto. Registered names, trademarks, etc., used in this publication, even without specific indication thereof, are not to be considered unprotected by law.

PRINTED IN THE UNITED STATES OF AMERICA

American Chemical Society
Library
1155 16th St., N.W.
Washington, D.C. 20036

ACS Symposium Series

M. Joan Comstock, *Series Editor*

1987 Advisory Board

Harvey W. Blanch
University of California—Berkeley

Alan Elzerman
Clemson University

John W. Finley
Nabisco Brands, Inc.

Marye Anne Fox
The University of Texas—Austin

Martin L. Gorbaty
Exxon Research and Engineering Co.

Roland F. Hirsch
U.S. Department of Energy

G. Wayne Ivie
USDA, Agricultural Research Service

Rudolph J. Marcus
Consultant, Computers & Chemistry Research

Vincent D. McGinniss
Battelle Columbus Laboratories

W. H. Norton
J. T. Baker Chemical Company

James C. Randall
Exxon Chemical Company

E. Reichmanis
AT&T Bell Laboratories

C. M. Roland
U.S. Naval Research Laboratory

W. D. Shults
Oak Ridge National Laboratory

Geoffrey K. Smith
Rohm & Haas Co.

Douglas B. Walters
National Institute of Environmental Health

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.fw001

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.fw001

Foreword

The ACS SYMPOSIUM SERIES was founded in 1974 to provide a medium for publishing symposia quickly in book form. The format of the Series parallels that of the continuing ADVANCES IN CHEMISTRY SERIES except that, in order to save time, the papers are not typeset but are reproduced as they are submitted by the authors in camera-ready form. Papers are reviewed under the supervision of the Editors with the assistance of the Series Advisory Board and are selected to maintain the integrity of the symposia; however, verbatim reproductions of previously published papers are not accepted. Both reviews and reports of research are acceptable, because symposia may embrace both types of presentation.

Preface

SOCIETY'S CONCERN ABOUT POLLUTANTS IN THE ENVIRONMENT requires pollution control and cleanup of hazardous waste sites. The 1986 Congressional reauthorization of the Comprehensive Environmental Response Compensation and Liability Act (CERCLA, or Superfund), similar initiatives in the Departments of Defense and Energy, and industry efforts may lead to expenditures on waste cleanup of $10-20 billion over the next five years. The problems are great, and the solutions are complex and expensive. Solutions require the cooperative effort of the public, government, industry, and the technical community. Yet, the methodology for achieving these solutions remains poorly understood, and advances in solving hazardous waste problems are rarely reported or are scattered in diverse literature sources.

The symposium on which this book is based was intended to publicize recent advances in the methodology of cleanup of sites contaminated with polychlorinated dibenzodioxins (PCDDs). PCDDs, and especially 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), are well-studied classes of chemicals that can serve as a model of approach to other hazardous chemical waste issues. Paul des Rosiers and I called on experts from many disciplines to define the potential problem, assess the risk, and describe the management of that risk. The multidisciplinary perspective covered the detection of the contaminant in the environment, its toxicology to living organisms, risk assessment through exposure routes, and elements in management of the risks. Because of the emotional nature of the dioxin issue, we strove to include speakers from many points of view. I have tried to maintain this balance in the book. I sincerely thank Paul des Rosiers for helping organize the symposium and all contributors.

It is my hope that this book can define a very complex problem and describe solutions. The examples of dioxin cleanup issues and procedures are offered to provide engineers, health scientists, regulators, lawyers, business people, and other concerned individuals with a methodology applicable to other hazardous chemicals. I believe that science can detect pollutants in the environment and estimate their potential health risk. Society as a whole, scientist and nonscientist, determines acceptable risk. Society also plays a major role in managing risk because we face many problems and have limited resources to deal with them all.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.pr001

In Solving Hazardous Waste Problems; Exner, J.;
ACS Symposium Series; American Chemical Society: Washington, DC, 1987.

I hope that we can address these important problems with sound information and a spirit of cooperation among the diverse interests, and I hope that we remember the fundamental intent: to solve hazardous waste problems.

JURGEN H. EXNER
International Technology Corporation
Martinez, CA 94553

December 1, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.pr001

In Solving Hazardous Waste Problems; Exner, J.;
ACS Symposium Series; American Chemical Society: Washington, DC, 1987.

Chapter 1

Perspective on Hazardous Waste Problems Related to Dioxins

Jurgen H. Exner

International Technology Corporation, 4585 Pacheco Boulevard, Martinez, CA 94553

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch001

Polychlorinated dibenzo-p-dioxins (PCDD) represent a highly visible, well-studied class of pollutants that are acutely toxic to animals. Information about these chemicals and experience with many environmental contamination episodes serve as a guide for solving hazardous waste problems caused by other pollutants. Solutions require the recognition of a problem, such as the widespread distribution of a pollutant in the environment, releases of pollutants from chemical processes, or their presence at hazardous waste sites. After concern about the presence of pollutants in the environment arises, the risk posed by chemicals such as dioxins must be assessed. Such risk assessment requires an understanding of the acute and chronic toxicity of the pollutant and knowledge of pathways by which humans and animals can be exposed to the chemical. Subsequent risk management involves social, political, legal, and economic factors that interact with potential technical options. The major issues relating to cleanup of dioxins in the environment are described from experiences at sites in the United States and Europe.

Hazardous waste problems permeate our society. Potential threats to human health from pollutants in the environment arouse great social and scientific controversy. Social concern has led to numerous environmental laws and regulations such as the Resource Conservation and Recovery Act of 1976 and amendments of 1984 (RCRA) and the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 and its 1986 amendments (CERCLA). RCRA addresses current waste by requiring stringent record keeping of all waste production, collection, treatment, storage, and disposal activities. CERCLA, or Superfund, addresses the cleanup of hazardous waste sites in the U.S.A. Out of about 25,000 sites that may exist in this country, about 25% have been examined, and about 1000 sites have now been placed on a priority list. During the last six years, studies on cleanup have begun at about 450 sites, but remedial action under Superfund has begun at only a few. In addition, cleanups by private industry have begun also. Progress has been impeded by a lack of understanding of the cleanup process and appropriate technologies, by uncertainties about risks and how to estimate and manage them, by political indecision, and by public fears. During this period, however, a methodology for addressing pollution problems has developed which

0097-6156/87/0338-0001$06.00/0
© 1987 American Chemical Society

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch001

continues to be refined. No other pollutant, with the possible exception of polychlorinated biphenyls (PCB), has contributed more to our understanding of waste issues than the polychlorinated dibenzo-p-dioxins (PCDD).

Dioxin as a Model in Waste Cleanup

PCDD represent a class of 75 compounds containing one to eight chlorine atoms on the dibenzo-p-dioxin moiety. These various dioxin isomers show remarkable differences in toxicity. At present, our concern focuses primarily on the compounds containing four, five, and six chlorine atoms at the 2,3,7,8- positions. This variability in toxicity contributes to the complexity of obtaining adequate scientific information.

Investigations of PCDD, and especially 2,3,7,8- tetrachloro-dibenzo-p-dioxin (2,3,7,8- TCDD), has led to major advances in environmental sampling and analysis, in toxicology, risk assessment, risk management, and waste treatment. Techniques used for dealing with dioxin contamination represent the most advanced procedures for hazardous waste cleanup. These advanced techniques were developed because of:

(1) Great public concern over well-publicized environmental contamination episodes such as the Agent Orange controversy, the Seveso release, and the Missouri episode,
(2) Very high acute animal toxicity and carcinogenicity, which has led to cleanup to unprecedented low residual concentrations, and
(3) Unwillingness by commercial waste disposers to accept dioxin-contaminated waste, a decision which led to testing of many novel dioxin treatment schemes.

Despite the attention paid to dioxin, these pollutants do not pose as acute a hazard as a large spill of a lethal gas (such as the Bhopal tragedy), or a burning disposal site. Rather, concern about dioxin focuses on long-term effects such as potential cancer formation or bioaccumulation in the food chain. Unlike many pollutants, PCDD are very insoluble in water and are not as likely to leach into groundwater as, for example, halogenated solvents. In contrast to air pollutants such as volatile hydrocarbons, PCDD have very low vapor pressure so that the vapor phase does not represent a major exposure route for dioxin. Finally, unlike pollution problems caused by chemicals produced for useful purposes, dioxin is a minute, unwanted byproduct of chlorophenol production and of combustion processes.

Problem Definition

Concern about dioxin's effect on human health can be traced to a number of industrial exposures, its recognition as a byproduct in the production of large-scale quantities of chlorophenols such as 2,4,5- trichlorophenol (TCP) and pentachlorophenol (PCP), the finding of birth defects in animals exposed to 2,3,7,8- TCDD, and the recognition in the 1970's of a more general environmental contamination due to emissions from combustion sources.

Dioxin and Chlorophenols. In 1954, workers in a chemical plant in

Hamburg, Germany, suffered skin eruptions similar to acne. This affliction can be caused by exposure to chlorinated organic chemicals. In this case, the exposure was traced to 2,3,7,8-TCDD, a byproduct formed in ppm concentration during the production of 2,4,5- TCP. TCP is an intermediate in the production of 2,4,5- trichlorophenoxyacetic acid (2,4,5- T), an herbicide with widespread past use. The presence of 2,4,5- T in Herbicide Orange, a defoliant used in the Vietnam War, introduced 0.1 - 50 ppm of 2,3,7,8- TCDD to the formulation known as Herbicide Orange (1). The finding that this contaminant led to birth defects in animals caused large political controversy and the cancellation of Herbicide Orange use. Ever since, the political emotions of the Vietnam War, the uncertainties about health effects on soldiers and civilians exposed to the spraying, and further contamination episodes have maintained the public's and news media's attention on dioxin. Similarly, great scientific effort has been expended on providing information to deal with health concerns and legal issues.

Seveso. In 1976, a chemical plant producing 2,4,5- TCP, an intermediate in the production of hexachlorophene, a bacteriocide, had an emergency pressure release (2). About 2 kg of TCDD plus additional amounts of TCP, ethylene glycol, and sodium hydroxide were released over a populated area of 1810 ha. Evacuation of hundreds of inhabitants and extensive remediation efforts maintained public attention over the next ten years (3).

Missouri. In 1971, a used oil transporter sprayed oil contaminated with TCDD on riding arenas and roads in Missouri for dust control. The death of about 60 horses and illnesses in children led to an investigation by the Centers for Disease Control (CDC). Health effects were attributed to TCDD in the oil in 1974 (4). However, governmental agencies responsible for action were under the mistaken impression that TCDD had a half-life in the environment of one year and, therefore, carried out only minimal cleanup. In 1982, this problem resurfaced when Times Beach, a community near St. Louis, was evacuated after TCDD concentrations above 1,000 ppb were detected in soil samples. Decisions on this evacuation were carried out under an atmosphere of scientific uncertainty, public emotion, and political pressures within U.S. EPA. At present, there are 43 contaminated sites in Missouri, some of which are being excavated and restored.

Hamburg, Germany. The Seveso experience focused renewed public attention on the Boehringer plant, the same chemical plant at which TCDD first was detected as a byproduct of 2,4,5- TCP. The presence of TCDD in 2,4,5- T led to a halt in production of the herbicide. Two years later, the remaining facility was closed because a range of PCDD and PCDF congeners was detected in a sophisticated recycle process involving thermal decomposition of lindane to trichlorobenzene. Cleanup of this $50 million facility involves building and equipment decontamination, soil and groundwater cleanup, and destruction of production residues.

New Jersey. Diamond Shamrock owned a 2,4,5- T production facility in Newark, New Jersey from 1951-1971. The facility was owned by

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch001

several different companies until 1983, when TCDD was detected at the plant site, in the surrounding neighborhood, and the adjoining river. Diamond Shamrock repurchased the property, cleaned up the neighborhood by excavating contaminated areas, and will begin remediation of the site after approval of plans by the State of New Jersey Department of Environmental Protection.

Pentachlorophenol (PCP) Users. PCP has been used as a wood preservative for many years. PCP contains octa-, hepta-, hexa-, and pentachlorinated dioxins. Since PCP was applied generally in an oil solution, spills and discharges from this operation have led to widespread migration of PCP and its impurities. The magnitude of these environmental discharges and possible remedial measures are being examined by U.S. EPA and responsible parties.

Sources From Combustion Processes. In 1977, PCDD and PCDF were detected in particulate emissions from municipal incinerators (5). Similar data were soon reported from around the world. In 1980, workers at Dow Chemical Company, using the most sensitive and specific analytical techniques of that time, detected a range of PCDD in residues from many combustion processes (6). These workers postulated that all combustion processes that contain chlorine sources produce PCDD. Although this postulate is not supported in all situations, dioxin emissions from combustion sources are major contributors to PCDD in the environment (7).

At present, municipal incinerators, hazardous waste incinerators, and metal smelters discharge from 1-100 ng/Nm^3 of PCDD, about 1-100 g/year per facility. The possibility of PCDD in exhaust emissions from cars has been raised recently, and the U.S. Environmental Protection Agency (EPA) has examined a range of other possible dioxin sources.

Dioxin in the Environment. 2,4,5- T manufacturing sites or areas where dioxin has been spilled contain from 1 ppb to 50 ppm of PCDD. Environmental soil background is in the ppt range. Buildings involved in chemical manufacture of 2,4,5- T contain about 1-50,000 ng/m^2. Buildings contaminated by PCB fires contain about 1-1,000 ng/m^2 of 2,3,7,8- TCDD equivalents. PCDD concentrations in ambient air are below the detection levels of several pg/m^3. However, continued increases in the sensitivity of analytical procedures are allowing detection of 0.2 pg/m^3 in some urban air samples.

Fish in the Great Lakes and Baltic contain 1-100 ppt of PCDD and the fat of snapping turtles accumulates about 400 ppt. In general, fat accumulates about 20 times the amount of dioxin present in the food source (8).

The fat of humans contains 1-20 ppt of TCDD. Other dioxin isomers are also present, including up to 900 ppt of octachlorodibenzodioxin (OCDD). A low level background of 1-10 ppt of 2,3,7,8- TCDD in the fat of mother's milk appears to be common in some parts of the industrialized world (8).

In considering environmental analysis for PCDD, great care must be taken that validated sampling and analytical methods are used. Also, appropriate numbers of samples must be included before conclusions about the importance of the findings can become firm.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch001

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch001

Clearly, then, PCDD occur in the environment, often at hot spots resulting from discharges from manufacture or spills, and PCDD accumulate in the food chain. Clearly, there is a potential exposure to PCDD in the environment. Since risk is a combination of exposure and of the inherent toxicity of the chemical, the toxicity of dioxins becomes the second criterion in defining whether these pollutants pose concern.

Toxicity. Experimental animals are exceedingly sensitive to TCDD. The LD50, the dose that kills half of a test group, for 2,3,7,8- TCDD is 0.6 µg/kg of body weight for male guinea pigs. Table I shows the acute toxicity of TCDD and the considerable species-dependence of TCDD (9). Reasons for these large differences may be different biochemical elimination or degradation pathways for different animals.

Human beings exhibit acute symptoms such as chloracne, effects on enzyme and nervous systems, and muscle and joint pains. Generally, these effects decrease after exposure is eliminated.

Lifetime daily dosages of 10-100 ng/kg of body weight of 2,3,7,8- TCDD administered to rats led to toxicity and carcinogenicity. At 1 ng/kg body weight per day, no effects were observed over two years (9).

Table I. Acute Toxicity of 2,3,7,8- TCDD During Oral Administration

Animal	LD50, µg/kg body weight
Guinea pig	0.6 - 2
Rabbit, male	115
Rat	22 - 45
Dog	300 - 3,000
Hamster	1,157 - 5,051

The second part of the problem definition phase shows clearly that PCDD should be a potential concern to humans and animals in the environment. The degree of concern that we as a society should have can be estimated by a formalized risk assessment process.

Risk Assessment

The presence of PCDD in the environment and the acute and chronic toxicity of 2,3,7,8- TCDD and similarly substituted congeners in animals raise concern. How serious should this concern be and what actions are warranted to relieve this concern? These questions are addressed by a risk assessment

procedure which attempts to formalize and make consistent a general intuitive process of assessing risk. The outcome of such an assessment often presents the image of a rigorously scientific determination of the size and certainty of risk. Before accepting these conclusions, it is imperative that the assumptions and uncertainties of such risk assessment are understood thoroughly. For example, it is common practice to use worst-case assumptions and upper-bound estimates and to combine these repeatedly. The final risk estimate, then, tends to amplify the real risk by several orders of magnitude. Presumably, this overestimation protects against currently unknown health effects such as adverse synergistic effects of pollutants. Despite these large uncertainties, regulatory agencies need a formalized, consistent, and site-specific process of risk assessment to define potential remedial actions. For each problem, we evaluate how humans can be exposed to a pollutant and what this exposure can do to their health.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch001

Exposure Routes. Dioxin can enter a person through:

- Dermal contact, absorption through skin,
- Inhalation, breathing of contaminated air, and
- Ingestion, eating contaminated materials such as soil, food, or drinking water contaminated by dioxin.

In assessing these three routes, the physical and chemical properties of PCDD in the environment are important. TCDD is not significantly degraded by microorganisms although certain fungi may be able to decompose the chemical over long periods of time. There are no demonstrated biological degradations of other PCDD. Under environmental conditions, PCDD are not hydrolyzed nor do they undergo reactions with environmental chemicals under environmental conditions. PCDD in solutions are photolyzed by sunlight. PCDD exhibit a very low vapor pressure but appear to photodegrade in the gas phase. PCDD adsorb readily on particulate matter, log K_{oc} = 6.4 for TCDD. This class of chemicals is very water insoluble, 10 - 20 ng/ℓ for TCDD, and partitions readily into organic matter, log K_{ow} = 6.6 for TCDD.

In most cases of environmental contamination by dioxin, migration of dioxin occurs on suspended particles. Thus, dioxin in soil can migrate via erosion processes, through air as wind-blown dust, or in the aqueous phase to rivers or groundwater. Dioxin in the vapor phase does not appear as a significant exposure source except at the point source of some combustion processes.

Leaching of pollutants to ground or surface waters is a major pathway of environmental contamination. Leaching of TCDD from soil containing 8-26,300 ng/g yielded aqueous leachates of 0.1 - 55.5 ng/ℓ (10) . These data suggest that TCDD movement in soil is slow. However, TCDD in Missouri has been detected at greater than 6 ft, in New Jersey at greater than 13 ft, and at even lower depths at Hamburg and Hyde Park. Examination of this information suggests that this downward migration is due to chromatographic desorption of TCDD by other organic pollutants such as chlorinated and aromatic solvents.

The major route by which humans are exposed to PCDD is ingestion of soil and food. Children in contaminated areas can

ingest contaminated soil. The magnitude of this potential exposure is the major factor in the 1 ppb action criterion set for contaminated sites in Missouri (11). Grazing cattle ingest soil and accumulate PCDD in fat. Fish and other aquatic animals accumulate dioxin from water, sediment, or food sources. Vegetables may contain small amounts of dioxin at the surface, possibly as dust particles trapped in the outer membranes (12). Accumulation of PCDD through the root system of plants appears to be very low, presumably because of the low water solubility of these compounds.

Additional exposure variables include duration of exposure, the absorbed fraction, the bioavailability, climatic conditions, and body weight. However, the major question in risk assessment, other than the exposure dose, addresses the effect of the pollutant on humans.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch001

Human Health Effects. Examination of exposure routes estimates the potential dose and length of exposure to people. The crucial question for any risk assessment, then, involves the effect that the exposure to the toxicant has on the human being. Several groups of workers and populations have been exposed to dioxin. Studies of workers exposed to industrial accidents, such as the 1949 Monsanto accident in Nitro, West Virginia, herbicide applicators in Scandinavia, U.S. military personnel who sprayed Agent Orange in Vietnam, and the population near Seveso showed chloracne to be the major common symptom of exposure to dioxin. A recent study of Quail Run, Missouri, residents suggests a lowering of the immune response system by exposed residents. Other concerns that have surfaced are possible increases of soft-tissue sarcomas and birth defects. However, since all of these studies involve epidemiologic investigations of groups of exposed people, these studies' conclusions are subject to scientific argument because of:

- group size, which affects the statistical validity of the results,
- the accuracy of the exposure dose and its duration, and
- appropriate controls, which incorporate uncertainty about lifestyle variables, smoking for example, which may affect the results.

Inconclusive or conflicting results from such studies are very disturbing to the public but reflect scientific uncertainty. Recent advances in PCDD analysis of human adipose and blood analyses which can detect and possibly differentiate between background and exposure offer an opportunity to resolve some of the concerns about exposure levels in such epidemiologic studies.

In the absence of human testing data, toxicity evaluations rely on extrapolating animal test data to humans. One major concern in this extrapolation is how similar animals such as rats are to humans. This question is especially critical for the case of dioxins, which show considerable toxicity variation between different species. The second major uncertainty in the extrapolation is the model that relates animal data to potential effects on humans. Of the several models that have been proposed, the linearized - multistage model is being used by U.S. EPA. This

model gives the highest plausible upper limit of risk and leads to lifetime acceptable daily intake of 0.0064-0.0572 pg/kg B.W. Dutch, Swiss, and Ontario workers, on the other hand, used threshold-based extrapolations to calculate acceptable lifetime daily dosages of 1-10 pg/kg B.W. of TCDD, because these agencies assume TCDD to be a cancer promoter rather than an initiator (9).

Nevertheless, PCDD, and especially 2,3,7,8- TCDD, exhibit high acute toxicity and many chronic effects in animals. It is prudent, therefore, to estimate acceptable exposure limits. In considering cleanup, it is imperative to establish acceptable levels of cleanliness. The risk assessment process tries to address this number, which has been the subject of much debate. How clean is clean indeed? In this author's opinion, we must address different criteria based on practical concerns. Thus, new emissions should be regulated to the most rigorous health-protective standards. For cleaning of old sites, a reasonable risk of one excess cancer in 100,000 - 1,000,000 may be a prudent compromise between resources needed to carry out the cleanup and potential risk.

This question of whether it is scientifically valid to derive the lifetime control limit by using threshold or non-threshold models defines what cleanup levels are proposed for a site. The action level proposed by CDC for residential soil in Missouri is 1 ppb, based on a series of exposure assumptions and on virtually safe doses for 10^{-6} cancer risk of 0.0276 pg/kg/day (11). If one assumes a different threshold - based model, as did Dutch, Swiss, German, and Canadian workers (9), one obtains maximum allowable daily intake of 1-10 pg/kg/day. If one uses the same exposure calculations as CDC, one could then accept 4-40 ppb in residential soil according to these allowable daily intakes. CDC and EPA have allowed 7 ppb as acceptable residual concentrations at an industrial site in New Jersey (13). At Seveso, cleanup levels were set at 45 ppt for nonagricultural soil and 7 ppt for agricultural soil (3). Office buildings in the United States have been cleaned to 3-25 ng/m^2 of 2,3,7,8-TCDD equivalents on surfaces and to less than 10 pg/m^3 in the office air (14). Appropriate cleanup levels continue to arouse great controversy, and one can achieve varying levels depending on the method one chooses for assessing the risk. In each case, the final cleanup level often represents an excruciating compromise between scientific, social, legal, and economic factors.

Table II illustrates an important concept in PCDD toxicity, the concept of toxicity equivalent factors. In situations where exposure to many different dioxin congeners can occur, for example, in buildings after PCB fires or in PCP contaminated wood treatment sites, acute and chronic animal toxicity data do not exist for the complex mixtures. By comparing a number of standard tests, regulatory agencies have developed toxicity equivalency for many dioxin and furan isomers (15). Although there continues scientific debate on the validity of this concept, and although the numbers may change with time, such an assessment seems a pragmatic approach to a difficult, time-consuming, and costly toxicological research program.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch001

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch001

Table II. PCDD Toxicity Equivalents

Compound	Relative Potency
2,3,7,8- TCDD	1
Other TCDD	0.01
1,2,3,7,8- PeCDD	0.5
1,2,3,6,7,8- HxCDD	0.04
1,2,3,7,8,9- HxCDD	0.04
1,2,3,4,7,8- HxCDD	0.04
Other HxCDD	0.0004
OCDD	0

Risk Management

Management of the risk posed by an environmental contaminant presents the final challenge in the process of solving hazardous waste problems. Once a concern has been identified and a potential risk has been estimated, reduction or removal of that risk must be addressed. The cleanup represents a fascinating political process that incorporates the public's perception of the risk, divergent social values and philosophies, legal constraints, economic considerations, and technical alternatives.

The public has heard about dioxin's high acute toxicity, its potent carcinogenicity, and its reproductive effects on animals. Dioxin has been intertwined with an unpopular war and claimed to cause malformed babies and health abnormalities. Dioxin led to the temporary evacuation of the residents of Seveso and the permanent relocation of about 2,000 residents in Times Beach, Missouri. In general, the public fears the presence of dioxin and follows complex scientific discussions of risk assessment with great difficulty. The general desire is to remove this unwanted intrusion and hazard. What the unfortunate victims of a dioxin contaminated area soon find out, however, is that emotional response to dioxin by other communities and environmental regulations prevents the simple solution of removing dioxin wastes to another site for disposal or treatment. At this time, no commercial landfills or incinerators accept dioxin-containing wastes. Rather, dioxin wastes must be stored on site, and thermal destruction processes must be approved by U.S. EPA. Similarly, destruction processes such as incineration, thermal desorption, photolysis, or chemical destruction require complex permitting procedures. The duration of these permit activities and the need

to prepare site-specific applications for mobile technologies are major obstacles in the development of new waste treatment technology for dioxins and other pollutants.

Legal obstacles also affect the course of cleanup actions. In many cases, a responsible party for a waste site is identified. Subsequent negotiations often result in carefully worded consent agreements specifying a rigorous course of action. Often, however, these agreements, which have judicial status, are signed before a site has been investigated completely. The work, then, tends to fulfill the requirements of the consent agreement rather than solving the problem that is uncovered during the investigation. In some cases, private or potential class-action suits prevent action by potentially responsible parties because taking action may be considered an admission of guilt. Other legal factors include regulatory restraints such as disposal restrictions or transport prohibitions.

Economic, technical, and social values are closely related in achieving solutions. Setting the cleanup standard includes social and political factors in addition to the risk assessment methodology. One factor, cost, relates directly to the cleanup standard. For example, excavating contaminated soil at one site in Missouri would cost 30% of envisioned cost if the cleanup standard were set at 10 ppb rather than at 1 ppb. Alternatively, excavation and thermal destruction of dioxin in soil is philosophically more attractive than containment and perpetual monitoring of the site. Yet, two aspects argue against destruction technology. First, the CERCLA guidelines for remediation at Superfund sites have, until very recently, focused on proven technology such as containment. Second, costs of destruction tend to be three to ten times higher than containment. In some cases, potential exposure to people can be eliminated by removing the people from the source of contamination. This solution may be the low cost solution, but it still requires maintenance of a waste site. Perhaps more important, the relocation solution destroys a community and has negative social consequences. Thus, social, economic, and technical considerations remain intertwined.

Cleanup Actions at Dioxin Contaminated Sites

During the last decade, various remedial actions were carried out on dioxin-contaminated wastes in the United States and Europe. Historically, building rubble from industrial accidents was used as fill or disposed in the ocean. In 1977, about 2.2 million gallons of Agent Orange and similar herbicide formulations was incinerated on the ship Vulcanus in the Pacific (1). This solution was simplified by the ability to transfer the waste to the ship at a remote location in the Pacific and by the large quantity of liquid waste, which made the incineration on ship economically possible. The destruction efficiency of this at-sea-incineration was about 99.9% according to the limits of analytical detection at that time.

Seveso. The release in 1976 at Seveso required cleanup of about 200,000 m^3 of contaminated soil material, mostly soil containing 2,3,7,8- TCDD in ppb concentrations, and about 41 drums of reactor residues containing ppm levels of dioxin. Italian authorities examined photochemical, chemical, and thermal processes as poten-

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch001

tial destruction or cleanup options (3). Construction of a large incinerator was prevented by cost and local opposition. The population feared that Seveso would become the center for treatment of Italy's waste after the immediate problem was resolved. Eventually, the contaminated soil was excavated and placed into two specially constructed landfills of 140,000 and 60,000 m^3 at a total cost of about $200 million (3). Two landfills were constructed because two municipalities were involved, and neither wanted to accept dioxin waste from the other. The drums, originally destined for a landfill in East Germany, eventually were found in a French butcher shop. In 1985, this waste was incinerated in a Ciba-Geigy incinerator in Basel, Switzerland, at an estimated cost of $2.5 million.

Verona, MO. During the 1970's, Syntex attempted to dispose of the tank residue from a hexachlorophene production carried out by a lessee, Nepacco, which had become defunct. Attempts at incineration in commercial incinerators in Minnesota and Texas were blocked by legal action. Disposal on the Vulcanus, in conjunction with the Agent Orange burn, faltered because of low volume, 4,300 gal, which made it economically unattractive to the ship's operators, and because of transportation difficulties in shipping the waste from Missouri to the Pacific. Syntex then used a specially developed process that could be applied on site, safely, within existing governmental regulations. The photochemical process reduced the TCDD concentration from 343 ppm to 0.2 ppm, less than was present in commercial 2,4,5- T, and destroyed over 7 kg of TCDD (16). Residues from this operation were originally scheduled for incineration in the EPA's mobile incinerator in 1982. At that time, the design of the mobile incinerator and existing regulatory concepts prevented incineration of dioxin wastes above 1 ppm. However, redesign and operation of the incinerator in 1985 proved these concerns invalid (17).

Missouri. In 1981, U.S. EPA Region 7 personnel began a renewed investigation into the fate of other waste material produced by Nepacco. Diligent investigatory work led them to over 100 sites on which dioxin-containing waste oil may have been spread for dust control. A large amount of preliminary sampling, which often used composites of relatively large areas, confirmed 43 contaminated sites, many of which were residential areas. These sites contained dioxin at concentrations up to 1,600 ppb at depths of up to several feet.

We began our evaluation of immediate response options shortly before the buyout of Times Beach. Table III lists some of the options that could be implemented in a short time frame and which would remove people from dioxin exposure at Quail Run Mobile Home Manor (A), and two other sites (18).

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch001

Table III. Options and Relative Costs for Immediate Response at Three Missouri Sites.

	Relative Cost, $		
Option	Site A	B	C
Cover and leave	1.0	1.0	1.0
Buy out and stabilize	6.2	12.5	1.1
Excavate, store on site, restore area	7.2	8.6	1.0
Excavate, transport to commercial landfill, restore	26.0	-	-
Excavate, commercial incinerator, restore	123	-	-

Examination of the relative costs for immediate action showed that excavation, on-site storage, and site restoration achieved the objective of separating the population from dioxin exposure as economically as buy out and relocation of residents.

In addition to immediate response options, we also examined a variety of longer-term options. The two most reasonable options were long-term storage of the contaminated soil in a centralized landfill or incineration in a specially designed, large-scale incinerator, with appropriate economics of scale (19, 20).

Despite much further evaluation of other treatment options by the Missouri Governor's Task Force (21) and other organizations, the concepts of storage or incineration remained the most promising. U.S. EPA and the State of Missouri faced great public opposition to a landfill such as the one at Seveso. Consequently, two paths of action emerged. EPA transported the mobile incinerator to Missouri and carried out a pilot test (17). This demonstration verified the expected capability of destroying dioxin-contaminated soil and sludge to 99.9999 %. Concurrently, a comprehensive excavation and storage plan was developed (22). This document included a safety and health plan, ambient air and industrial hygiene sampling, an excavation procedure for removing soil at 6-inch intervals, and a statistical sampling and analysis plan to validate that a cleaned area meets the required degree of cleanliness of less than 1 ppb dioxin (23). A community relations plan was an integral part of both projects, and it was this concern for public involvement that helped achieve useful action in Missouri. At this time, soil has been excavated from the Cashel, Sullins, and Quail Run sites. The soil has been placed in bags which are stored in buildings erected at each contiguous site. The sites have been restored and are being inhabited.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch001

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch001

The actions taken in Missouri can serve as a model for responding to large environmental contamination in populated areas. However, decisions at other sites must consider site-specific factors such as potential groundwater contamination or land use, i.e., is it an industrial area, a residential area, or farm or grazing land. Also, the quantity of pollutant must have a bearing on decisions because long-term effects on the environment can be more serious for large quantities than for minimal amounts. Table IV summarizes some remediations and site-specific factors affecting the decisions.

Table IV. Site-Specific Factors in Remediation

Site	Dioxin Concentration ppb	Quantity Estimate	Geographic Features
Jacksonville, AR	0 - 1,200[a]	250 g[a]	Plant site adjacent to residential
	15,000 - 50,000	264 lbs	area, fractured bedrock
Newark, NJ	0 - 20,000[a]	10 - 100 lbs	Plant site in heavily populated area near river
Niagara Falls, NY	0 - 1,000[a]	100 - 2,000 lbs.	Closed landfills containing large amounts of organic residues, fractured bedrock
Missouri sites	0 - 1,600[a]	40 - 70 lbs	Mostly residential, fractured limestone under clay

a. In soil

The Arkansas and New Jersey sites became contaminated because of production of 2,4 - D and 2,4,5 - T. The estimated 264 lbs of dioxin at Jacksonville are contained in drummed TCP residues. The Love Canal and Hyde Park landfills in New York probably contain larger quantities of dioxin than any sites other than the Georgswerder landfill in Germany. Because these landfills also contain large quantities of TCP and chlorinated solvents, leaching from these sites into ground or surface water is probable. All the sites exhibit an underground geology of fractured bedrock

which makes leaching of dioxin possible in the presence of solvents. Such subsurface migration into the environment is least likely for those Missouri sites that contain only TCDD.

Remedial action at sites other than Missouri has been affected by site-specific factors and legal factors. Consent agreements at the New York landfills and the Arkansas site allowed temporary control measures such as containment, capping, and monitoring. In New Jersey, the proximity to a river, the high population density, and the magnitude of the problem make it difficult to work out reasonable destruction options such as excavation and incineration. Yet, barrier walls, caps, and groundwater pumping and treating require indefinite monitoring and maintenance, and the land serves no further useful function. So, the decision on which cleanup option to pursue becomes exceedingly complex.

Current Cleanup Options for Dioxin Wastes. Governmental regulations and dioxin's negative image currently prevent off-site disposal or treatment of dioxin-contaminated wastes. In response to a need to destroy dioxin wastes, a number of novel process adaptations have been proposed and tested. These processes include incineration, physical-chemical, and biological processes. In addition, on-site containment, storage, and monitoring remain economic alternatives.

Microbial destruction of dioxin congeners remains an elusive goal. At present, small conversions of 2,3,7,8- TCDD in solution by fungi have been achieved in very small - scale laboratory experiments. Extrapolation of this concept to organic wastes or contaminated soil, on a large scale, faces formidable technical obstacles. This concept, as presently envisioned, appears about five years away from being realized in practical applications.

Over the last five years, U.S. EPA has supported research on a chemical dehalogenation process for destroying PCB and dioxin. Nucleophilic displacement of aromatic halides by alkali-metal polyglycoxides occurs readily at elevated temperatures. This reaction readily dechlorinates PCB in mineral oil at 100° and proceeds similarly with 1,2,3,4- TCDD in organic solvents. The addition of dipolar, aprotic solvents such as dimethyl sulfoxide increases the reaction rate and allows dechlorination reactions in dry systems at ambient conditions. Initial tests of this concept for soil decontamination at Shenandoah Stables, Missouri, failed because of high moisture content of the soil and low temperatures. The process was used successfully in the summer of 1986 to remove penta-, hexa-, hepta-, and octachlorodibenzodioxins from 9,000 gal of fuel oil that also contained 2 % PCP. This operation, however, left about 30 drums (ca. 1,500 gal) of polyglycol/DMSO/water/KCL/KOH waste, the ultimate disposal of which is uncertain. The process is being tested further on PCB - contaminated soil which is excavated and placed into a reactor system. The major questions on this process remain economics, residual solvents in treated oil or soil, and ultimate disposal of the polyglycol residues. The final disposal of chemical treatment residues presents a similar problem as residues from photochemical destruction of dioxin.

Incineration of dioxin wastes is the most versatile destruction process of those presently available. The mobile incinerator treated a combination of soil, sludge, and solvents

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch001

successfully. However, costs associated with such a small research-oriented unit were greater than $2,000 per ton, so that considerable efforts are under way to improve the mobile incinerator and to explore other thermal destruction processes. One such method, the Huber reactor, has been tested at Missouri and Gulfport, Mississippi. Although these tests destroyed dioxin in dioxin - contaminated soil, two aspects of the process remain a concern. First, the soil must be dried to less than 2 % moisture and reduced in size to about 100 microns. Second, in part because of these pre-treatment steps, costs for this process are estimated in the range of $700/ton.

Two very promising alternatives for treating dioxin - contaminated soil are the Shirco process and the thermal desorption method. The Shirco process places contaminated soil on a moving grate. The soil is heated by infrared heaters or by burners, and vapors are destroyed in a secondary combustion chamber. This process has been demonstrated on a small scale at the Times Beach test facility and will be tested further under various private and governmental sponsorships.

The thermal desorption process, sponsored by U.S. Air Force, has been demonstrated on a small scale, 100 - 200 lbs/hr, at Gulfport, Mississippi, and Johnston Island. The process consists of indirect heating of contaminated soil in a rotating cylinder. The desorbed vapors can be destroyed in a secondary combustion chamber or collected in a condensing or scrubber system. The demonstration unit incorporated a scrubber and photolysis system rather than a combustion unit for the following reasons:

- The desorption part was the operation of interest since combustion had been demonstrated by U.S. EPA, and
- a physical-chemical treatment was presumed to be easier to permit than an incineration device.

Considerations of regulatory obstacles, then, defined the path of technology development in this instance.

Future Developments. Cleanup of sites contaminated by pollutants should accelerate after the recent reauthorization of Superfund. Methodology for investigating sites must improve, particularly sampling and analysis procedures that are effective and efficient. Risk evaluation and risk management continue to evolve as society gains more experience. Since these risk evaluations require considerable judgment about public will, legal constraints, and scientific facts, decisions will vary from country to country, from state to state, and possibly from community to community. In developing this experience base, we must remember to protect the environment and people by placing stringent restrictions on new or continuing emissions and by incorporating risk and benefit in assessing the necessary cleanup level for existing, old sites. In the technical area, immediate advances can be made in improving the efficiency of incineration processes by understanding temperature and residence time relationships in the desorption and secondary combustion chamber. For example, is the two second and 1,200° C requirement for secondary combustion necessary for dioxin wastes? Developments in improving incinerator operations can reduce also

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch001

emissions from these thermal processes. Research on in situ treatment of contaminated wastes offers clear public and economic advantages. Carefully designed, understood, and applied biological systems offer promise for achieving in-place treatment of dioxin and other pollutants at low capital costs over the next ten years.

Literature Cited

1. Young, A.L.; Calcogni, J.A.; Thalken, C.E.; Tremblay, G.W.; "The Toxicology, Environmental Fate and Human Risk of Herbicide Orange and Its Associated Dioxin," National Technical Information Service AD/A-062143, Springfield, VA, 1978.
2. Carreri, V. in "Dioxin: Toxicological and Chemical Aspects," Cattabeni, F.; Cavallaro, A.; Galli, G., Eds., Spectrum Publications: Jamaica, NY, 1978, p.1.
3. Fortunati, G.V., in "Chlorinated Dioxins and Dibenzofurans in Perspective," Rappe, C.; Choudhary, G.; Keith, L.H., Eds., Lewis Publishers Inc.: Chelsea, MI, 1986, p.541.
4. Carter, C.D.; Kimbrough, R.D.; Liddle, J.A.; Cline, R.F.; Zack, M.M.; Barthel, W.F.; Koehler, R.E.; Philipps, P.E., Science 1975, 188, 738.
5. Olie, K.; Vermeulen, P.L.; Hutzinger, O., Chemosphere, 1977, 8, 455
6. Bumb, R.R. et al.; Science 1980, 210, 385.
7. Crummett, W.B.; Townsend, D.I.; Chemosphere 1984, 13, 777.
8. Rappe, C. in "Solving Hazardous Waste Problems: Dioxins," Exner, J.H., Ed.; ACS SYMPOSIUM SERIES No.____, American Chemical Society: Washington, D.C., 1987, p___.
9. Kociba, R.J., in "Solving Hazardous Waste Problems: Dioxins," Exner, J.H., Ed.; ACS SYMPOSIUM SERIES No._____; American Chemical Society: Washington, D.C., 1987; pp____ - _____.
10. Jackson, D.R.; Roulier, M.H.; Grotta, H.M.; Rust, S.W.; Warner, J.S.; in "Chlorinated Dioxins and Dibenzofurans in Perspective," Rappe, C.; Choudhary, G.; Keith, LH., Eds., Lewis Publishers: Chelsea, MI, 1986, p.185.
11. Kimbrough, R.; Falk, H.; Stehr, P.; Fries, G., J. Tox. Environmental Health 1984, 14, p.47.
12. Facchetti, S.; Balasso, A., Fichtner, C.; Frare, G.; Leoni, A.; Mauri, C.; Vasconi, M.; in "Chlorinated Dioxins and Dibenzofurans in Perspective," Rappe, C.; Choudhary, G.; Keith, L.H.; Eds., Lewis Publishers: Chelsea, MI, 1986, p.225.
13. E. Noble, personal communication, 1985.
14. Gravitz, N.; Fan, A.; Neutra, R.R.; California Department of Health Services, "Interim Guidelines for Acceptable Exposure Levels in Office Settings Contaminated with PCB and PCB Combustion Products," 1983.
15. Bellin, J.S.; Barnes, D.G.; Toxic and Industrial Health, 1985, 1, 235.
16. Exner, J.H., Johnson, J.D., Ivins, O.D., Wass, M.W., Miller, R.A., in "Detoxication of Hazardous Waste," Exner, J.H., Ed., Butterworth: Stoneham, MA, 1982, p.267.
17. Kleopfer, R.D.; Hazel, R.H.; Freestone, F.; desRosiers, P.E., in "Chlorinated Dioxins and Dibenzofurans in Perspective,"

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch001

Rappe, C.; Choudhary, G.; Keith, L.H.; Eds.; Lewis Publishers: Chelsea, MI, 1986, p.511.

18. Exner, J.H.; Erikson, D.G.; Cibulskis, R.; Keffer, W.D.; in "1984 Hazardous Material Spills Conference Proceedings," Ludwigson, J., Ed., Government Institutes, Inc., Rockville, MD, 1984, p.245.
19. Exner, J.H.; Erikson, D.; "Quick-Response Engineering Assessment of Removal Options for Quail Run Mobile Home Park, MO, Dioxin Contamination," Draft Report to U.S. EPA Region 7, Kansas City, KS, September 1, 1983.
20. Exner, J.H.; Alperin, E.S.; Groen, A.; Morren, C.E.; Kalcevic, V.; Cudahy, J.J.; Pitts, D.M.; in "Chlorinated Dioxins and Dibenzofurans in the Total Environment," Keith, L.H.; Rappe, C.; Choudhary, G., Eds., Butterworth: Stoneham, MA, 1985, p.47.
21. Finch, J.A. Jr. et al., "Final Report of the Missouri Dioxin Task Force," State of MO, October 31, 1983.
22. Keffer, W.U.; Wurtz, S.; Newbore, G.; Howard, D.; Exner, J.H.; "Quail Run Hazard Mitigation Plan," U.S. EPA Region 7 Report, Kansas City, KS, 1984.
23. Exner, J.H.; Keffer, W.J.; Gilbert, R.O.; Kinnison, R.R.; Hazardous Waste and Hazardous Materials, 1985, 2, p.503.

RECEIVED January 14, 1987

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch001

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch002

Chapter 2

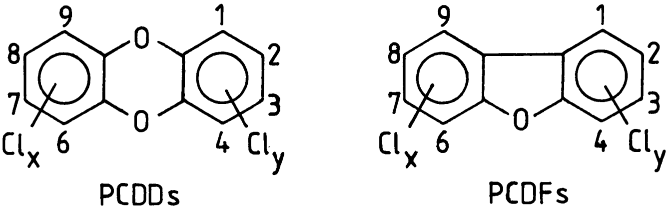
Global Distribution of Polychlorinated Dioxins and Dibenzofurans

Christoffer Rappe

Department of Organic Chemistry, University of Umeå, S-901 87 Umeå, Sweden

Polychlorinated dioxins and dibenzofurans have been identified in technical products and pesticides, most of which are not very widely used today. Other sources are incinerators of various types like MSW incinerators, but also hazardous waste incinerators and industrial incinerators. PCDDs and PCDFs have also been identified in exhausts from cars running on leaded gasoline with halogenated additives. Background levels of PCDDs and PCDFs have been identified in fish and other aquatic organisms from the Great Lakes and the Baltic Sea, and also in human adipose tissue samples from USA, Canada, Sweden, Japan and Vietnam as well as in samples of breast milk from Sweden, Denmark, West Germany, the Netherlands, Yugoslavia and Vietnam. The isomeric pattern in all these biological samples is very similar.

The polychlorinated dibenzodioxins (PCDDs) and dibenzofurans are two groups of compounds that exhibit similar chemical and physical properties, the chemical structures are given below:



0097-6156/87/0338-0020$06.00/0
© 1987 American Chemical Society

The number of chlorine atoms in these compounds can vary between one and eight (x+y = 1-8) to produce 75 PCDD and 135 PCDF positional isomers. A few of these have strong toxic effects, specifically 2,3,7,8-tetra-CDD and the other 2,3,7,8-substituted Cl_4, Cl_5 and Cl_6 PCDD and PCDF isomers, in all 12 congeners, see Table I.

Table I. The Most Toxic PCDD and PCDF Isomers

PCDDs	PCDFs
2,3,7,8-Tetra-CDD	2,3,7,8-Tetra-CDF
1,2,3,7,8-Penta-CDD	1,2,3,7,8-Penta-CDF
1,2,3,6,7,8-Hexa-CDD	2,3,4,7,8-Penta-CDF
1,2,3,7,8,9-Hexa-CDD	1,2,3,6,7,8-Hexa-CDF
1,2,3,4,7,8-Hexa-CDD	1,2,3,7,8,9-Hexa-CDF
	1,2,3,4,7,8-Hexa-CDF
	2,3,4,6,7,8-Hexa-CDF

In the present presentation the primary interest is focused on reports discussing the contamination and distribution of all PCDDs and PCDFs and not only 2,3,7,8-tetra-CDD in various matrices in the environment and in the general population. This enables the possibility to discuss congener profiles as well as patterns of individual isomers.

Sources of PCDDs and PCDFs

Commercial and technical products. During the 1960's and 1970's most of the interest was focused on the PCDD and PCDF contamination in technical products like 2,4,5-T and other chlorophenoxy acids, pentachlorophenol and other chlorinated phenols, and PCB. The levels were found to vary from batch to batch, but the analyses were mostly non-isomer specific. The major contaminant in 2,4,5-T was 2,3,7,8--tetra-CDD, normally in the lower ppm-range or less (1). In general, technical pentachlorophenols were found to be the most contaminated products, levels were reported in the range of 1000 ppm, mainly for octa-CDD, but also a multitude of lower chlorinated congeners (1).

As a result of the increasing concern over PCDD and PCDF impurities, efforts were made to reduce the levels in the commercial products. These operations, however, often resulted in an increased volume of contaminated waste streams. Very, very few analyses were made or reported on PCDD and PCDF levels in these waste streams, but numerous outbreaks of chloracne and other skin problems should have indicated a potential problem. These waste streams are now recognized as the source of numerous environmental problems.

In many industrialized countries the use of products contaminated by PCDDs and PCDFs is nowadays highly reduced. For instance 2,4,5-T, earlier a major herbicide in most countries, is now only produced and used in New Zealand and PCB is being phased out as dielectric fluid in transformers and capacitors in most countries during the 1980's.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch002

Municipal incinerators. In 1977-1978 two European groups reported that a series of PCDDs and PCDFs could be found in fly ash samples collected in the electrostatic precipitator of municipal solid waste (MSW) incinerators (2,3). Buser and Bosshardt (3) studied fly ash from a MSW incinerator and an industrial heating facility, both in Switzerland. In the former the total level of PCDDs was reported to be 0.2 ppm and of PCDFs 0.1 ppm. In the industrial incinerator the levels were 0.6 ppm and 0.3 ppm, respectively. Since then a great number of reports from Europe, U.S.A., Canada, and Asia have confirmed the original findings. However, for many years the interest was focused on the analysis of fly ash samples and not on the analysis of the total emissions, which could be explained by sampling problems.

Most of the analyses of samples from incinerators have been done using non-validated and non-isomer specific sampling and analytical methods. Recent studies show the presence of a multitude of PCDD and PCDF congeners, in fact all isomers seem to be present (4). Moreover, a striking similarity in the isomeric pattern of PCDDs and PCDFs was found between samples from different incinerators (4,5).

Various models have been used to convert a multitude of levels of more or less toxic PCDDs and PCDFs into a more simple expression like "TCDD equivalents" or "toxic equivalents" (6). In Sweden and in this report the approach discussed by Eadon et al. (7) has been used.

Emissions from MSW incinerators operating under good conditions are in the range of 1-100 ng TCDD equivalents /Nm^3 (8, 9) resulting in total annual emissions of 1-100 g of TCDD equivalents from a normal size MSW incinerator; 50 000 - 200 000 tons/year. The major chlorine source in the MSW are plastic material like PVC and bleached and unbleached paper (10). New technologies are now available to reduce the emissions.

Hazardous waste incinerators. Hazardous waste incinerators have also been the object of public concern. However, available data indicate the emissions from such incinerators operating under good conditions are of the same magnitude as emissions from MSW incinerators. The isomeric patterns of PCDDs and PCDFs are similar to those reported from MSW incinerators (4,5).

Industrial processes. Recent work in our group has identified industrial high-temperature processes like copper smelters and electrical arcing furnaces in steel mills as sources of environmental contamination of PCDDs and PCDFs (8). The copper smelter used scrap copper containing PVC-coated wires and cords. In the steel mill a high portion of the alloys or stainless steel was recycled. This recycled material was contaminated by PVC or polychlorinated paraffins (8). The emission from these sources seem to be of the same magnitude as emissions from MSW incinerators. However, due to a larger number of units, the total emissions from these industrial processes could exceed the emissions from MSW incinerators. The emissions from coal fired power plants, wood and peat burners seem to be very low (per Nm^3) but the very large number of units could make a significant total contribution (8).

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch002

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch002

Automobiles. Very recently Ballschmiter et al. identified a series of PCDDs and PCDFs in used motor oil from automobiles (11). These authors also reported on a striking similarity in the isomeric pattern of the PCDFs found in this matrix and in samples from MSW incinerators (11). Ballschmiter et al. suggest chlorinated additives in the motor oil or in the gasoline as possible sources for the PCDDs and PCDFs, but no quantitative data is given (amount per km). Also very recently Marklund et al. reported on a study where real car exhausts were analyzed for PCDDs and PCDFs (12). The test cars were collected in two groups

1. Cars equipped with a catalytic converter using unleaded gasoline with no halogenated scavengers.
2. Cars with no catalytic converter using leaded gasoline (0.15 g/l) and a dichloroethane scavenger (0.1 g/l).

Before the test runs the motor oil was exchanged in all cars.

No PCDDs and PCDFs could be identified in the cars using the unleaded gasoline, while the emission average from the cars running on leaded gasoline was found to be 30-540 pg/km of TCDD equivalents (Eadon). It was assumed that the chlorinated scavenger (dichloroethane) was the precursor to the PCDDs and PCDFs formed. It was estimated that the total amount of PCDDs and PCDFs from cars in Sweden using leaded gasoline with halogenated scavengers is in the range of 10-100 g TCDD equivalents/year (12).

An extreme good similarity was reported for the isomeric pattern of the tetra- and penta-CDFs found in automobile exhausts and in emissions from MSW incinerators (12) and steel mills, see Figure 1.

Levels of PCDDs and PCDFs in Environmental Samples

Soil and sediments. A great number of analyses have been performed on soil and sediment samples, preferentially from "hot spots" where levels of PCDDs and PCDFs, mainly 2,3,7,8-tetra-CDD, as high as ppm have been reported, e.g. Seveso, Times Beach and Love Canal. In a few studies background levels of PCDDs and PCDFs in soil and sediment samples are reported. However, in most of these studies non--validated and non-isomer specific analytical methods are used. These studies indicate background levels in the low ppt range, but in general no specific isomers are reported.

Olie (13) reported on the analysis of 14 soil samples collected from 11 locations in previously sprayed habitats of southern Viet Nam during the period 1980-81. 2,3,7,8-Tetra-CDD was identified in four of these samples in levels of 16-31 ppt.

Recent data (5) using validated (^{13}C) and isomer specific analytical methods indicate that new sediments from the archipelago of Stockholm, Sweden, contain ppt or sub-ppt levels of a variety of PCDD and PCDF isomers. The isomeric profiles found in these sediments are very similar to those found in samples from MSW and industrial incinerators and car exhausts.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch002

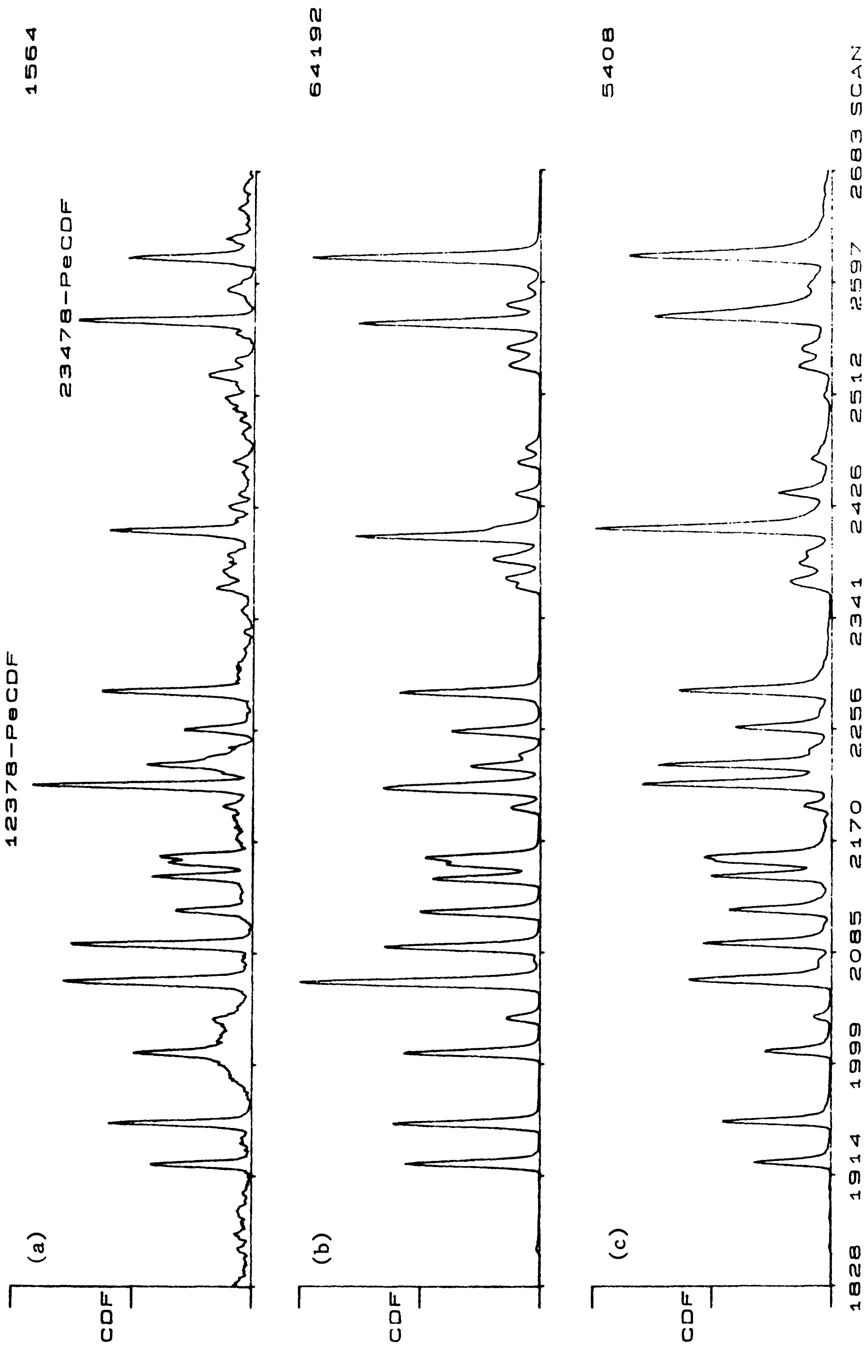


Figure 1. Fragmentograms of penta-CDFs in samples from (a) car exhausts, (b) MSW incinerator, and (c) dust from baghouse in steel mill.

Air and particulate. Due to sampling and analytical problems very few data are available on the levels of PCDDs and PCDFs in normal urban air. Samples of air particulates from Washington, D.C., and St. Louis, MO, USA have been analyzed by Crummett et al. (14) and Czuczwa and Hites (15). Crummett et al. (14) report on isomer specific analyses for tetra-CDDs and hexa-CDDs while Czuczwa and Hites (15) only report a congener profile and do not discuss any specific isomers. In both studies octa-CDD was found to be a major constituent, reported in levels of 150-200 ppb. The level of 2,3,7,8-tetra--CDD was 5-50 ppt and of 2,3,7,8-tetra-CDF was 100-380 ppt (14).

Recent studies using pre-spiked (^{13}C) air samples and isomer specific analytical methods (5) indicate that sub pg/m^3 levels of a variety of PCDDs and PCDFs can be found in air sampled in an industrialized area. The isomeric patterns found in these samples were very similar to those found in samples from MSW and industrial incinerators, car exhausts and also in new sediments (5).

Also very recently Oehme et al. (16) reported on a study where urban air was investigated by high-volume sampling of 1000 m^3 of air using a glass fiber filter and polyurethane foam as collection media. Measurable amounts of a large number of PCDDs and PCDFs were found in cities and industrialized regions, while the levels in remote areas were lower. The highest levels were in the order of 5-10 pg/m^3 for the sum of all PCDDs and PCDFs. In suburban areas the levels were lower by a factor of 5-10 (16).

Aquatic organisms. In the late 1970s and early 1980s analytical methods were developed to analyze for 2,3,7,8-tetra-CDD as well as other PCDDs and PCDFs (Table 1) in biological samples. Due to the high toxicity of these compounds the detection levels should be in the order of pg/g (ppt) or lower. Such analyses are complicated and expensive and up to now only a limited number of analyses are reported, mainly from U.S.A., Canada and Sweden.

Mitchum et al. (17) reported on levels up to 400 ppt of 2,3,7,8-tetra-CDD in a waterway associated with the production of 2,4,5-T. Levels ranging from 4-695 ppt of 2,3,7,8-tetra-CDD were found for the edible portion of various fishes from Saginaw Bay, U.S.A., the highest concentrations were detected in bottom-feeding catfish and carp. In all 36 samples were analyzed and 26 of these were reported to have detectable amounts: > 5-10 ppt of 2,3,7,8--tetra-CDD. Ten samples contained concentrations greater than 40 ppt (18).

Rappe et al. identified a series of tetra to octa-CDFs in fat samples of a snapping turtle from Hudson River and gray seal from Baltic Sea. The total levels of PCDFs in these samples were 3 ng/g and 40 pg/g, respectively. In both samples the major PCDFs consisted of the most toxic isomers: 2,3,7,8-tetra-, 2,3,4,7,8-penta- and 1,2,3,4,7,8- and 1,2,3,6,7,8- hexa-CDF (19).

Fish and other aquatic animals from the Great Lakes have been analyzed and the data indicate low background levels (1-300 ppt) of a series of PCDDs and PCDFs. Norstrom et al. have analyzed pooled samples of herring gull eggs collected in 1982 from various parts of the Great Lakes. In all samples 2,3,7,8-tetra-CDD was found in levels ranging from 9 to 90 pg/g (20). In another study Stalling et al. (21) were not able to identify measurable levels of tetra-CDDs

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch002

and other PCDDs in fish samples from Lake Superior (the detection level was 2-5 pg/g). The difference in these two studies could be explained by the migration of the herring gulls during the winter. On the contrary a series of PCDFs could be identified in the Lake Superior fish samples (15-40 ppt), indicating a more widespread background levels for the PCDFs than for the PCDDs. Stalling et al. also reported on the analysis of fish samples from Lakes Michigan, Huron and Ontario. Total levels of PCDFs were found to be 12-290 ppt (21). The toxic 2,3,7,8-substituted PCDDs and PCDFs were present in all samples, the highest levels being found in samples from Lake Huron, Lake Ontario and Tittabawasee River. The residue pattern found in the fish and locally high levels suggest a strong influence by point source discharges (21). The Baltic Sea is another system studied which could be of interest because this is a system without any known point sources. Some relevant data from the Baltic Sea are collected in Table II (22).

Table II. Levels of PCDDs and PCDFs Found in Samples from the Baltic Sea (22)

		Seal pg/g	Salmon pg/g	Guillemot pg/g	Herring pg/g
2,3,7,8-	TCDD	28	6	17	0.6
1,2,3,7,8-	PeCDD	60	22	26	ND
1,2,3,4,7,8-	HxCDD	2.9	0.8	0.9	ND
1,2,3,6,7,8-	HxCDD	69	6.6	18	ND
1,2,3,7,8,9-	HxCDD	3.6	0.2	1.6	ND
1,2,3,4,6,7,9-	HpCDD	0.4	0.5	ND	ND
1,2,3,4,6,7,8-	HpCDD	1.2	0.9	0.6	ND
	OCDD	2.5	4.3	0.2	ND
2,3,7,8-	TCDF	12	62	0.2	3.9
1,2,3,7,8-	PeCDF	3.7	24	1.8	1.2
2,3,4,7,8-	PeCDF	125	82	97	5.6
1,2,3,4,7,8-	HxCDF	1.1	9.1	4.7	0.6
1,2,3,6,7,8-	HxCDF	2.4	6.3	10	0.4
2,3,4,6,7,8-	HxCDF	1.5	5.1	4.5	0.7
1,2,3,4,6,7,8-	HpCDF	0.2	41	0.5	0.5
1,2,3,4,6,7,9-	HpCDF	ND	0.6	ND	ND
1,2,3,4,6,8,9-	HpCDF	ND	2.2	ND	0.2
1,2,3,4,7,8,9-	HpCDF	ND	0.9	T	ND
	OCDF	1.8	61	T	ND

ND = not detected < 0.1 pg/g
T = trace

An interesting observation is that in the majority of the aquatic samples only the toxic 2,3,7,8-substituted congeners are found, compare Tables I and II. Crustaceans seem to be an exception from this general behaviour; Norstrom et al. (23) reported in 1985 that hepatopancreas from crabs from the Canadian Pacific coast

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch002

contain other congeners, e.g. 1,2,4,7,8-penta-CDD and 1,2,3,6,7,9-/ /1,2,3,6,8,9-hexa-CDD. Recent analyses of hepatopancreas and meat from crabs and lobsters collected at the U.S. Atlantic Coast and the Swedish West Coast confirm this observation (5). The levels in the hepatopancreas were found to be 10-100 times higher than the levels found in the meat. Consequently crustaceans are good signal organisms to study sources of environmental pollution of PCDDs and PCDFs, the highest levels of pollutants are found in the hepatopancreas.

Terrestrial animals. At the time of the accident in Seveso, Italy in 1976 more than 81 000 animals were inhabiting the contaminated zones. The main part were rabbits (25 000) and poultry and other small animals (55 500), but also 349 cattle, 233 pigs, 49 horses, 31 sheeps and 49 goats were in the zones. Many of these animals died, others were killed. A large number of these animals were analyzed for 2,3,7,8-TCDD by a method with a detection level at 250 pg/g (24). The results are collected in Table III.

Table III. TCDD analyses on liver of farm animals from contaminated zones and surrounding areas (1976-1979) (24)

Animal	Number of samples	TCDD-containing samples (>250 pg/g)	TCDD maximum level ng/g
Rabbits	698	433	633
Poultry	83	35	24
Cattle	43	21	94
Horses	12	2	88
Pigs	13	0	-
Goats	25	17	1

Nygren et al. (22) analyzed bovine samples: fat, liver and milk and identified the same 2,3,7,8-substituted PCDDs and PCDFs as were found in the aquatic samples. However, the levels were lower and normally close to the detection limit, see Table IV. This indicates that the background levels are higher in the aquatic environment than in the terrestrial environment, which is in agreement with earlier experience for substances such as DDT and PCB.

Levels of PCDDs and PCDFs in Human Samples

Adipose tissue. In April 1984 Rappe et al. (25) reported on a series of the toxic 2,3,7,8-substituted PCDDs and PCDFs in samples of human adipose tissue from Northern Sweden. A series of reports presented at the dioxin conferences in Ottawa, Canada in October 1984, Miami, Fl, U.S.A. in April 1985 and in Bayreuth, West Germany in September 1985 confirmed these observations, and it is clearly shown that there is a background of PCDDs and PCDF in the general population in the industrialized part of the world. In Table V are

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch002

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch002

Table IV. Levels of PCDDs and PCDFs found in bovine fat, bovine liver, bovine milk and cream samples (22)

		Cow fat Sweden pg/g	Cow liver Sweden pg/g	Cow fat Scotland pg/g	Milk Denmark pg/g fat	Milk Scotland pg/g fat	Cream Sweden pg/g fat
2,3,7,8-	TCDD	ND	ND	NA	NA	NA	NA
1,2,3,7,8-	PeCDD	ND	ND	2	T	5	ND
1,2,3,4,7,8-	HxCDD	ND	ND	1	ND	3	ND
1,2,3,6,7,8-	HxCDD	ND	ND	2	6	6	18
1,2,3,7,8,9-	HxCDD	ND	ND	ND	6	2	ND
1,2,3,4,6,7,8-	HpCDD	3	4	3	9	2	42
	OCDD	9	10	3	< 86	3	16
2,3,7,8-	TCDF	ND	ND	ND	ND	ND	2
2,3,4,7,8-	PeCDF	ND	ND	5	3	8	4
1,2,3,4,7,8-	HxCDF	ND	ND	1	ND	3	3
1,2,3,6,7,8-	HxCDF	ND	ND	1	8	2	T
2,3,4,6,7,8-	HxCDF	ND	ND	1	4	2	3
1,2,3,4,6,7,8-	HpCDF	ND	ND	2	< 7	3	6
	OCDF	ND	ND	ND	< 7	< 15	< 20

ND = not detected (< 1.0 pg/g)
NA = not analyzed
T = trace

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch002

Table V. Levels of PCDDs and PCDFs in Human Adipose Tissue (ppt on wet weight)

Isomer		Sweden n=31 (22)	USA/NY n=8 (26)	Canada n=46 (26)	Japan n=13 (27)	N Viet Nam n=9 (26)	S Viet Nam n=15 (26)
2,3,7,8-	TCDD	3	7.2	6.4	9	< 2	18
1,2,3,7,8-	PeCDD	10	11.1	10	15	< 2	9.1
1,2,3,6,7,8-	HxCDD	15	95.6	81	70	5.6	57
1,2,3,7,8,9-	HxCDD	4	NA	NA	12	NA	NA
1,2,3,4,6,7,8-	HpCDD	97	164	135	77	17	121
	OCDD	414	707	850	230	52	900
2,3,7,8-	TCDF	3.9	NA	NA	9	NA	NA
2,3,4,7,8-	PeCDF	54	14.3	15	25	7.2	12
1,2,3,4,7,8-	HxCDF	6	NA	NA	15	NA	NA
1,2,3,6,7,8-	HxCDF	5	31.3	16	14	7.7	33
2,3,4,6,7,8-	HxCDF	2	NA	NA	8	NA	NA
1,2,3,4,6,7,8-	HpCDF	11	16.5	30	NA	4.2	17
	OCDF	4	NA	NA	NA	NA	NA

ND = not detected (< 1.0 pg/g)
NA = not analyzed

collected some relevant data from U.S.A., Canada, Sweden and Japan. For comparison, data from North and South Viet Nam are also included in this table (22, 26, 27). Data from other countries seem not to be available.

It is interesting to note the similarity in isomers, levels, and isomeric pattern in these samples collected from the industrialized countries on three continents. The PCDD profile shows increasing levels with increasing number of chlorine atoms, the level of OCDD is 400-900 ppt while the PCDF profile has a maximum for 2,3,4,7,8-penta-CDF or 1,2,3,6,7,8-hexa-CDF. The difference in levels found between samples from North and South Viet Nam can be explained by the difference in industrial activities between the two parts of the country.

Breast milk. In April 1984 Rappe et al. also reported on the analyses of five samples of breast milk from West Germany (25). In January 1985 Rappe reported on the analyses of four samples of breast milk from the Umeå region in northern Sweden (28), and in May Nygren et al. reported on additional single samples from Denmark and Viet Nam. In September 1985 Fuerst et al. reported on the analyses of 53 samples of breast milk from West Germany (29). The data from these investigations are collected in Table VI together with data from pooled samples of breast milk from the Netherlands and Yugoslavia (5). Low levels of PCDDs and PCDFs were found in all samples from all these countries. Discussing the specific isomers and levels we find great similarities between adipose and breast milk samples as shown in Tables V and VI (4,5,9).

Conclusions

Available data, although from very few countries, indicate a general low background for 2,3,7,8-substituted PCDDs and PCDFs in the environment, especially for aquatic organisms and also in the general population.

In most biological samples only the most toxic PCDD and PCDF isomers were found, see Tables I-VI. In addition, two Cl_7 and two Cl_8 congeners were also found. An exception from this general behaviour is the crustaceans, where a multitude of isomers could be identified.

A poor correlation was observed between the isomeric distribution found in environmental and human samples on one side and the potential sources on the other. This indicates a combination of sources e.g. incineration and technical products in addition to environmental and biological degradation. Of special interest here is the observation of 1,2,3,7,8-penta-CDD in all biological samples. This isomer is found in all samples from incinerators and also in low levels in a few technical pentachlorophenol formulations (5).

It is of great importance that ultratrace analyses of PCDDs and PCDFs are generated by representative sampling and validated analytical methods. Greater efforts should be devoted to interlaboratory studies, e.g. Albro et al. (30). Several of the analytical methods used in this study generated data of high quality. However, other methods were not so safe, a large portion of the reported data were false positives or false negatives.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch002

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch002

Table VI. Levels of PCDDs and PCDFs Found in Breast Milk (ppt on fat weight)

		Sweden n=4 (28)	W. Germany n=5 (25)	W. Germany n=53 (29)	Viet Nam n=1* (22)	Denmark n=1 (22)	Netherlands n=3 (5)	Yugoslavia n=2 (5)
2,3,7,8-	TCDD	0.6	1.9	ND	< 0.5	NA	9.7	< 1.0
1,2,3,7,8-	PeCDD	6.5	12.9	11.0	7.0	31	44	5.5
1,2,3,4,7,8-	HxCDD	2.5	NI[a]	8.6	ND	13	25	3.5
1,2,3,6,7,8-	HxCDD	19	NI[a]	32.9	50	97	251	15
1,2,3,7,8,9-	HxCDD	6.3	NI[a]	6.4	24	32	23	ND
1,2,3,4,6,7,8-	HpCDD	59.5	72.8	48.8	150	174	130	28
	OCDD	302	434	143	754	328	744	106
2,3,7,8-	TCDF	4.2	5.4	1.7	9.4	4.0	2.8	< 1.0
1,2,3,7,8-	PeCDF	< 1	< 1	2.0	< 1	4.5	ND	ND
2,3,4,7,8-	PeCDF	21.3	36.4	21.1	21	31	79	25
1,2,3,4,7,8-	HxCDF	4.7	NI[b]	8.3	15.0	13	8.9	3.7
1,2,3,6,7,8-	HxCDF	3.4	NI[b]	6.9	11.0	52	10.3	3.6
2,3,4,6,7,8-	HxCDF	1.4	NI[b]	3.2	4.2	11	6.4	1.3
1,2,3,4,6,7,8-	HpCDF	7.4	9.2	7.4	23.0	46	39	ND
	OCDF	3.2	2.4	27.0	46.0	ND	ND	ND

NI = not isomer specific
ND = not detected
NA = not analyzed

a) Total level of hexa-CDDs = 23.4 ppt
b) Total level of hexa-CDFs = 26.0 ppt

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch002

Literature Cited

1. Rappe, C. Env. Sci. Technol. 1984, 16, 78 A 90 A.
2. Olie, K.; Vermeulen, P.L.; Hutzinger, O. Chemosphere 1977, 8, 455-459.
3. Buser, H.R.; Bosshardt, H.-P. Mitt. Geb. Lebensm. Hyg. 1978, 69, 191-199.
4. Rappe, C.; Marklund, S.; Kjeller, L.O.; Bergqvist, P.-A. and Hansson, M. In "Chlorinated Dioxins and Dibenzofurans in the Total Environment". Keith, L.H.; Rappe, C.; Choudhary, G., Eds.; Butterworth: Stoneham, MA, 1984; Vol. II p. 401.
5. Unpublished data: University of Umeå, Sweden.
6. Bellin, J.S.; Barnes, D.G. Toxicology and Idustrial Health 1985, 1, 235-248.
7. Eadon, G.; Aldous, K.; Hilker, P.; O'Keefe, P.; Smith, R. "Chemical data on air samples from the Binghamton State Office Building", 1983. Memo from Center for Lab Research, New York State Department of Health, Albany, NY 12201, 7/7/83.
8. Marklund, S.; Kjeller, L.-O.; Hansson, M.; Tysklind, M.; Rappe, C.; Ryan, C.; Collazo, H.; Dougherty, R. In "Chlorinated Dioxins and Dibenzofurans in the Total Environment". Rappe, C.; Choudhary, G.; Keith, L., Eds.; Lewis Publishers, 1986, Vol. III p. 79.
9. "PCDD and PCDF Emissions from Incinerators for Municipal Sewage Sludge and Solid Waste-Evaluation of Human Exposure". WHO Regional Office for Europe, Copenhagen, Denmark, IPC/CEH 003/m 06 1986.
10. Churney, K.L.; Ledford, A.E.; Bruce, S.S.; Domalski, E.S. U.S. Department of Commerce Report NBSIR 85-3213, 1985.
11. Ballschmiter, K.; Buchert, H.; Niemczyk, R.; Munder, A.; Swerev, M. Chemosphere. In press.
12. Marklund, S.; Rappe, C.; Tysklind, M.; Egebäck, K.-E. Chemosphere. In press.
13. Olie, K. In "Herbicides in War. The Long-term Ecological and Human Consequences". Westing, A.H.; Ed. SIPRI, Stockholm, Sweden, 1984, p. 173.
14. Crummett, W.B.; Nestrick, T.J.; Lamparski, L.L. In "Dioxins in the Environment". Kamrin, M.A.; Rodgers, P.W. Eds. Hemisphere Publishing, New York, N.Y. 1985, 75-76.
15. Czuczwa, J.M.; Hites, R.A. Env. Sci. Technol. 1986, 20, 195-200.
16. Oehme, M.; Manø, S.; Mikalsen, A.; Kirschmer, P. Chemosphere, 1986, 15, 607-617.
17. Mitchum, R.K.; Moler, G.F.; Korfmacher, W.A. Anal. Chem. 1980, 52, 2278-2282.
18. Harless, R.L.; Lewis, R.G. In "Chlorinated Dioxins and Related Compounds. Impact on the Environment". Hutzinger, O.; Frei, R. W.; Merian, E.; Pocchiari, F.; Eds., Pergamon Press, Oxford, 1982, p. 25.
19. Rappe, C.; Buser, H.R.; Stalling, D.L.; Smith, L.M.; Dougherty, R.C. Nature, 1981, 292, 524-526.

20. Norstrom, R.J.; Hallett, D.J.; Simon, M.; Mulvihill, M.J. In "Chlorinated Dioxins and Related Compounds. Impact on the Environment". Hutzinger, O.; Frei, R.W.; Merian, E.; Pocchiari, F.; Eds., Pergamon Press, Oxford, 1982, p. 173.
21. Stalling, D.L.; Smith, L.M.; Petty, J.D.; Hogan, J.W.; Johnsson, J.L.; Rappe, C.; Buser, H.R. In "Human and Environmental Risks of Chlorinated Dioxins and Related Compounds". Tucker, R.E.; Young, A.L.; Gray, A.P., Eds., Plenum Press, New York and London, 1983, p. 221.
22. Nygren, M.; Rappe, C.; Lindström, G.; Hansson, M.; Bergqvist, P.-A.; Marklund, S.; Domellöf, L.; Hardell, L.; Olsson, M. In "Chlorinated Dioxins and Dibenzofurans in the Total Environment". Rappe, C.; Choudhary, G.; Keith, L.; Eds., Lewis Publishers, 1986, Vol. III. p. 79.
23. Norstrom, R.J.; Simon, M.; Rappe, C.; Bergqvist, P.-A. Chemosphere. In Press.
24. Pocchiari, F.; Di Domenico, A.; Silano, V.; Zapponi, G. In "Accidental Exposure to Dioxins. Human Health Aspects". Coulston, F.; Pocchiari, F., Eds., Academic Press, New York and London, 1983, p. 5.
25. Rappe, C.; Bergqvist, P.-A.; Hansson, M.; Kjeller, L.-O.; Lindström, G.; Marklund, S.; Nygren, M. Banbury Report 18: Biological Mechanisms of Dioxin Action. Cold Spring Harbor Laboratory, NY, U.S.A. 1984, p 17-25.
26. Schecter, A.; Gasiewicz, T.; Ryan, J.J.; Gross, M.; Weerasinghe, W.C.A.; Constable, J. Presentation at this conference.
27. Ono, M.; Wakimoto, T.; Tatsukawa, R.; Masuda, Y. Chemosphere. In Press.
28. Rappe, C. In "Organo Halogen Compounds in Human Milk and Related Hazards" Report on a WHO Consultation. WHO Regional Office for Europe, Copenhagen, Denmark, IPC/CEH 501/m 05. August 1985.
29. Fuerst, P.; Meemken, H.-A.; Groebel, W. Chemosphere. In Press.
30. Albro, P.W.; Crummett, W.B.; Dupuy, A.E.; Gross, M.L.; Hansson, M.; Harless, R.L.; Hileman, F.D.; Hilker, D.; Jason, C.; Johnson, J.L.; Lamparski, L.L.; Lau, B. P.-Y.; McDaniel, D.D.; Meehan, J.L.; Nestrick, T.J.; Nygren, M.; O'Keefe, P.; Peters, T.L.; Rappe, C.; Ryan, J.J.; Smith, L.M.; Stalling, D.L.; Weerasinge, N.C.A.; Wendling, J.M. Anal. Chem. 1985, 27, 2717-2725.

RECEIVED November 25, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch002

Chapter 3

National Dioxin Study

Paul E. des Rosiers

U.S. Environmental Protection Agency (RD-681), Washington, DC 20460

This report presents the results of EPA's investigation of potential 2,3,7,8-TCDD (dioxin) contamination. The study represents a two-year, nationwide, multi-media evaluation initiated at the request of the U.S. Congress in 1983. The majority of dioxin contamination at Tier 1, 1a, 2 and 2a sites remained on-site. At sites where concentrated 2,4,5-TCP production wastes were stored or disposed, 2,3,7,8-TCDD concentrations were as high as 356 ppm. At most sites, however, 2,3,7,8-TCDD levels in soil were usually in the ppb range. In fish samples from nearby lakes and streams, 2,3,7,8-TCDD was measured in terms of ppt. Only two Tier 3 sites were extensively contaminated and comprised large facilities handling 2,4,5-T, 2,4,5-TP and 2,4,5-TCP with extent of contamination limited to one or two soil samples above 1 ppb. CDDs and CDFs were present in stack emissions from all sources tested in Tier 4 and most, but not all, of the combustion source categories reported in the literature. CDD and CDF emissions from some sources have estimated risks to the most exposed individual of 10^{-5} or more; these sources include a secondary copper smelting facility, a sewage sludge incinerator, and some municipal incinerators. At Tier 5 pastureland, rice field, and sugarcane sites, 2,3,7,8-TCDD levels in contaminated soils ranged from 0.6-564 ppt, with 67 percent below 5 ppt; levels in fish filets were between 8 and 23 ppt. At the three Tier 6 regionally selected sites, none was extensively contaminated. At one site, however, groundwater contamination was found at the 0.07-0.10 ppt level in three samples. The Tier 7 investigation established the prevalence of 2,3,7,8-TCDD in the environment: 2,3,7,8-TCDD was detected infrequently and at very low levels in background soil samples with the highest level being 11.2 ppt.

This chapter not subject to U.S. copyright.
Published 1987, American Chemical Society

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003

Basis of Concern

Numerous incidents of dioxin contamination have resulted in a high level of public awareness and concern. This concern carries over into present efforts to implement cleanup actions and conduct disposal operations.

Among the best known incidents was exposure of U.S. forces to Agent Orange in Viet Nam. A defoliant, Agent Orange, was contaminated with 2,3,7,8-TCDD. A large number of suits was filed against the manufacturers of these chemicals. These cases have now been settled out-of-court, thus the issue of cause and effect was not addressed.

In Seveso, Italy, an industrial accident involving 2,4,5-trichlorophenol (2,4,5-TCP)/hexachlorophene (HCP) manufacture resulted in widespread, low-level 2,3,7,8-TCDD contamination. Effects included evacuation of segments of the community, animal deaths, extensive cleanup efforts, and treatment of over 100 cases of chloracne alleged to be associated with the accident.

In Times Beach, Missouri, waste oil from 2,4,5-TCP/HCP manufacture, contaminated with 2,3,7,8-TCDD, was used to control dust on roads. The result was the well-known government 'buy-out' of the town. Use of waste oil at several Missouri horse arenas resulted in acute human health effects and the death of sixty-five horses and several small animals. Subsequent use of the horse arena materials as fill at building construction sites resulted in further cleanup problems.

2,3,7,8-TCDD and other chemical contamination of adjacent land and water as a result of industrial waste disposal at the Love Canal, 102nd Street, and Hyde Park sites in Niagara Falls, New York, posed health risks. 2,3,7,8-TCDD contamination was also found to be extensive at the Vertac facility in Jacksonville, Arkansas, both on- and offsite.

In 1979, investigations at a municipal incinerator in Hempstead, New York, led to the discovery that dioxins (CDDs) and furans (CDFs) were being emitted during the combustion process.

In 1980, a PCB transformer fire in the basement of the State Office Building in Binghamton, New York, resulted in distribution of soot containing high levels of CDDs and CDFs, including the 2,3,7,8-isomers, throughout the building. Cleanup costs to date exceed the original construction cost of the building.

In 1981, reports from Canada demonstrated the presence of 2,3,7,8-TCDD in Great Lakes' fish. These reports coincided with reports of contamination of fish in several U.S. rivers, notably the Titabawassee River in Michigan.

The National Dioxin Strategy

At the time of the Congressional request for a study, the Environmental Protection Agency (EPA) was in the midst of responding to contamination in Missouri and other locations. EPA also had rulemaking proceedings underway to address various aspects of the dioxin problem.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003

EPA's National Dioxin Study had three objectives:

- To study the extent of dioxin contamination and the associated risks to humans and the environment;
- To implement or compel necessary cleanup action at contaminated sites; and
- To evaluate further both disposal alternatives to alleviate current problems and regulatory alternatives to prevent future contamination.

On December 15, 1983, EPA issued a "national dioxin strategy" for investigating, identifying, and cleaning up sites contaminated by dioxin (1). Within the framework of this strategy was a plan that called for research to be conducted on the technical feasibility and economics of alternative methods for disposal and destruction of wastes and soils contaminated by dioxin.

To implement this strategy, EPA established seven categories (tiers) of investigation and study ranging from the most probable contamination to the least. These are:

Tier 1. 2,4,5-TCP production sites and associated waste disposal sites.

Tier 2. Sites and associated waste disposal sites where 2,4,5-TCP was used as feedstock for pesticidal products.

Tier 3. Sites and associated waste disposal sites where 2,4,5-TCP and its derivatives were formulated into herbicidal products.

Tier 4. Combustion sources such as municipal and hazardous waste incinerators, PCB transformer/capacitor fires, reactivation furnaces for spent granular activated carbon, boilers burning PCBs or pentachlorophenol (PCP)-treated wood, etc.

Tier 5. Sites where herbicides derived from 2,4,5-TCP have been and are being used on a commercial basis such as rights-of-way, rice fields, forests, certain aquatic areas, and pastureland.

Tier 6. Organic chemical and pesticide manufacturing facilities where improper quality control of certain production processes would have resulted in the formation of 2,3,7,8-TCDD.

Tier 7. Control sites where contamination of 2,3,7,8-TCDD is not suspected. These are to be compared with known contaminated sites to form a background level for the strategy studies.

Toxicity Equivalence

While the primary focus of this study was on contamination associated with 2,3,7,8-TCDD, EPA was also concerned with human exposure from other isomers of chlorinated dibenzo-p-dioxin and dibenzofuran.

EPA (2) and others have developed a number of weighting schemes to compare the toxicities of various chlorinated dioxin and furan isomers to that of 2,3,7,8-TCDD, the most studied, and generally considered to be the most toxic isomer. Toxicity

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003

Equivalence Factor (TEF) approaches express the toxicity of CDD/CDF mixtures in terms of "2,3,7,8-TCDD equivalents." This allows comparison of the toxicity of mixtures for purposes of risk assessment and remedial action planning. EPA's TEFs for isomers and homologs of CDD/CDF are shown in Table 1.

With the exception of 2,3,7,8-TCDD, the 2,3,7,8-substituted HxCDDs and 2,3,7,8-TCDF, these TEFs are not based on the results of major animal (reproductive, carcinogenic) studies. Generally, TEFs are based on estimates of the relative toxicity from in vitro tests whose relationship to the chronic effects of concern is largely presumptive. Nevertheless, studies on systemic effects continue to reinforce the view that the short-term assays provide important fundamental information on the toxicity of the CDDs/CDFs.

Tiers 1 and 2/1a and 2a, Production and Disposal Sites

Tier 1 comprises 2,4,5-TCP production sites and associated waste disposal sites (Tier 1a); whereas Tier 2 represents sites where 2,4,5-TCP was employed as a precursor in the manufacture of pesticides such as 2,4,5-T, 2,4,5-TP (Silvex), or hexachlorophene, and associated waste disposal sites (Tier 2a).

EPA used a three-phased approach. First, information was collected to identify these sites. EPA then used its enforcement authorities to verify production facilities and associated waste disposal sites. As needed, EPA collected additional data from site visits and employee interviews.

Next, EPA screened the sites through field testing to determine actual 2,3,7,8-TCDD contamination. EPA employed a biased sampling approach. Instead of taking samples randomly, EPA sampled in the locations most likely to be contaminated with dioxin (i.e., loading, storage, or production areas). If offsite migration of contaminated material was suspected, EPA sampled outside the facility boundary. Quality assurance/quality control protocols, following Superfund-authorized procedures, were developed to ensure that all data generated would be of known quality.

Finally, if a level of 2,3,7,8-TCDD contamination requiring a cleanup response (i.e., >1 ppb) was identified, potentially responsible parties (PRPs) were encouraged to take appropriate response action. If necessary, response activities were performed under Superfund.

All activities were coordinated with state and local authorities as well as other federal agencies such as the Centers for Disease Control (CDC), the National Institute for Occupational Safety and Health (NIOSH), the Food and Drug Administration (FDA), and the Federal Emergency Management Agency (FEMA). Community relations played an important role in all activities conducted during the identification, screening, and cleanup of dioxin-contaminated sites.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003

Table 1. Relative Potency Factors Used in Estimating 2,3,7,8-TCDD Equivalents

Compound(s)	Relative Potency Factor or TEF[a]
2,3,7,8-TCDD	1.0
Other TCDDs[b]	0.01
Penta-CDDs	0.5
Hexa-CDDs	0.04
Hepta-CDDs	0.001
Octa-CDD	0.000
2,3,7,8-TCDD	0.1
Other TCDFs[b]	0.001
Penta CDFs	0.1
Hexa-CDF	0.01
Hepta-CDFs	0.001
Octa-CDF	0.000

[a] Relative potency factor for the most toxic 2,3,7,8-substituted isomer.
TEF = toxic equivalence factor = relative toxicity assigned.
CDD or CDF concentration x TEF = 2,3,7,8-TCDD equivalents.

[b] In situations where 2,3,7,8-TCDD or -TCDF was not chemically analyzed in the sample, then TCDDs and TCDFs will have a relative potency factor of 1.0 and 0.1, respectively.

Results. Originally, about 450 Tier 1 and 2 sites (50 production sites and about 400 associated waste disposal sites) were expected. After systematic searching, the number of sites has been more clearly defined. In total, there were 99 dioxin sites in Tiers 1 and 2. Table 2 shows a breakdown of sites by tier (3):

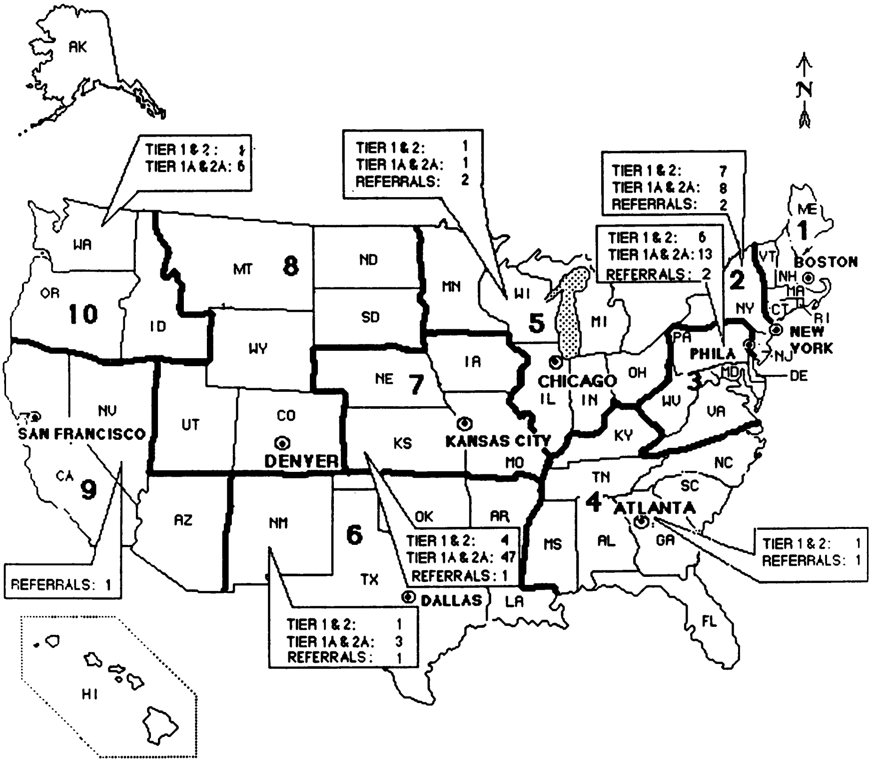
Table 2. Dioxin Sites by Tier

Tier	Number of Sites
1	11
1a	53
2	9
2a	26

Tier 1 and 2 sites are located in six of the ten EPA Regions. Figure 1 is a graphic representation of dioxin-contaminated site locations by Region (3).

Seventeen dioxin sites are on the current Superfund National Priorities List (NPL); four more have been proposed in Updates 2 and 3 to the NPL. Most of these NPL sites, such as Love Canal

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003



TOTALS

TIER 1 & 2: 20
TIER 1A & 2A: 79
REFERRALS: 10

Figure 1. Regional Distribution of Dioxin Tier 1/Tier 2 Sites

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003

in Niagara Falls, New York, are on the NPL for chemical contamination problems in addition to dioxin. The 21 sites on the NPL or proposed NPL (Updates 2 or 3) are (3):

Cashel Residence	Fenton, MO
Conservation Chemical	Kansas City, MO (Update 3)
Diamond Shamrock	Newark, NJ
Drake Chemical	Lock Haven, PA
Fike Chemicals	Nitro, WV
Hooker Chemical (Hyde Park)	Niagara Falls, NY
Hooker Chemical (Love Canal)	Niagara Falls, NY
Hooker Chemical (S-Area)	Niagara Falls, NY
Hooker Chemical (102nd St.)	Niagara Falls, NY
Minker Residence	Imperial, MO
Moyers Landfill	Collegeville, PA
NIES	Furley, KS (Update 2)
Quail Run	Gray Summit, MO (Update 2)
Rohm & Haas	Bristol, PA (Update 3)
Romaine Creek	Imperial, MO
Shenandoah Stables	Moscow Mills, MO
Stout Residence	Imperial, MO
Sullins Residence	Fenton, MO
Times Beach	Times Beach, MO
Vertac	Jacksonville, AR
Western Processing	Kent, WA

Under the National Dioxin Strategy, sites found to be contaminated in Tiers 3 through 7 are referred to the Office of Solid Waste and Emergency Response (OSWER) for possible Superfund (CERCLA) action. These sites are then considered along with the sites in Tiers 1 and 2 and all other hazardous waste sites managed under the Superfund program.

Additionally, the EPA Regions are responding to several sites, not identified through the National Dioxin Strategy, where dioxin may be present. Following is a list of the ten dioxin referral sites:

Bogle Dioxin Site	Alexandria, VA
Chem All, Inc. (Sonford Chemical)	Port Neches, TX
Chemspray Inc. (Agrisystems)	Belleglade, FL
E.T.M. Enterprises (Parsons Chemical Works, Inc.)	Grand Ledge, MI
Farmingdale Garden Labs	Farmingdale, NY
Riverdale Chemical Co.	Chicago Heights, IL
Smith-Douglas	Norfolk, VA
Stauffer Chemical (Montrose Chemical)	Henderson, NV
Union Carbide Agricultural Products	St. Joseph, MO
W.A. Cleary	Somerset, NJ

Of the 99 Tier 1 and 2 production and waste disposal sites, ten are classified as requiring "no further action" under the Strategy based on sampling results indicating very low or undetectable levels of 2,3,7,8-TCDD. These sites are:

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003

Calgon Corporation	Pasadena, TX
Drake Chemical	Lock Haven, PA
Eastman Kodak	Rochester, NY
Georges Creek	Poca, WV
GROWS	Morrisville, PA
Millmaster Onyx	Berkeley Heights, NJ
Moyers Landfill	Collegeville, PA
Rhone-Poulenc	Portland, OR
South Charleston Landfill	South Charleston, WV
Union Carbide	South Charleston, WV

Of those sites where dioxin was detected at levels of concern, the most common materials contaminated were soil and dust. At certain sites, production waste, contaminated equipment, and contaminated building materials were found. As expected, these sites were generally the production facilities of Tiers 1 and 2 and those waste disposal sites where intact waste or scrapped equipment was stored or disposed. The majority of dioxin contamination at Tier 1, 1a, 2 and 2a sites remained on-site. Furthermore, the 2,3,7,8-TCDD was usually confined to areas where waste materials were stored, loaded, processed, or, in the case of 28 Missouri sites, sprayed on roads to control dust. In seven cases, offsite migration at levels of concern has been confirmed. They are:

Diamond Shamrock	Newark, NJ
Brady Metals	Newark, NJ
Love Canal	Niagara Falls, NY
102nd Street	Niagara Falls, NY
Hyde Park	Niagara Falls, NY
Vertac	Jacksonville, AR
Bliss Tank Property	Frontenac, MO

Levels of Contamination. Where 2,3,7,8-TCDD was detected, levels generally were highest in the vicinity of actual waste handling operations (e.g., processing, loading, and storage). At sites where concentrated 2,4,5-TCP production wastes were stored or disposed, 2,3,7,8-TCDD concentrations were as high as 356 ppm. At most sites, however, 2,3,7,8-TCDD levels in soil were usually in the ppb range. In fish samples from nearby lakes and streams, 2,3,7,8-TCDD was measured in terms of ppt.

Cleanup Activities. Cleanup actions have focused on reducing potential exposure through inhalation or ingestion of dioxin-contaminated soils or dust. Many techniques have been developed and/or evaluated by the EPA Office of Research and Development (4) and have been employed by OSWER and others in cleanup activities.

Tier 3, Formulators

Tier 3 consists of facilities and associated waste disposal sites where 2,4,5-TCP and its derivatives were formulated into

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003

pesticidal products. Generally, these products are herbicides, insecticides, fungicides, and germicides:

2,4,5-trichlorophenol (2,4,5-TCP)	fungicide, bactericide
2,4,5-trichlorophenoxyacetic acid (2,4,5-T)	plant hormone; herbicide; defoliant
silvex (2,4,5-TP)	herbicide; plant growth regulator
erbon	herbicide
ronnel	insecticide
hexachlorophene	topical anti-infective (restricted); germicidal soaps; veterinary medicine
isobac 20	topical anti-infective (restricted); germicidal soaps; veterinary medicine

Figure 2 depicts sampling locations nationally.

Findings. EPA estimates that approximately 8 percent of the 312 facilities identified in the FATES database are contaminated above 1 ppb in soil or above detection limits of approximately 10 ppq - 1 ppt in other media. At dioxin-contaminated sites, the extent of contamination was usually limited to one or two soil samples with concentrations of 2,3,7,8-TCDD above 1 ppb. Only two Tier 3 sites were extensively contaminated and comprised large handlers of 2,4,5-T, 2,4,5-TP, or 2,4,5-TCP (5).

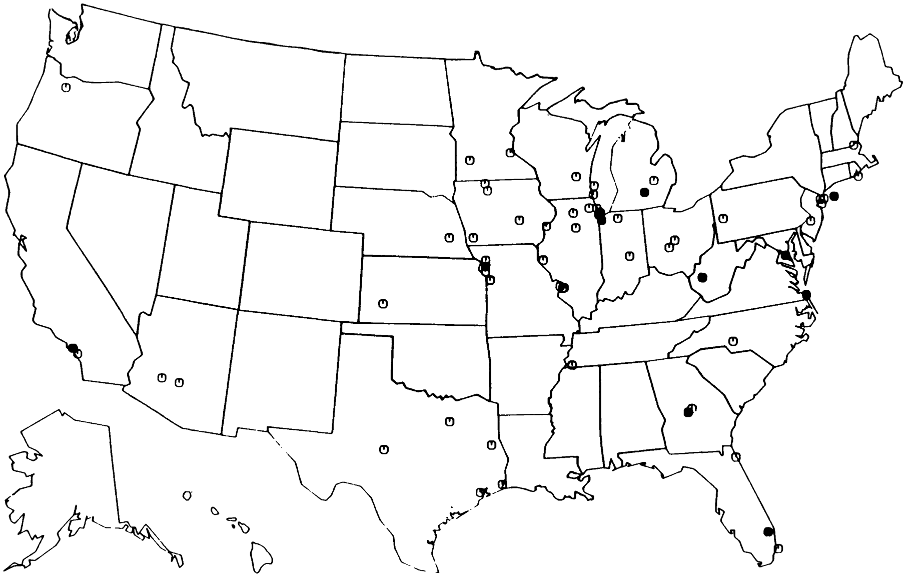
Tier 4, Combustion Sources

This section summarizes the findings of Tier 4, the portion of the study dealing with combustion sources. Tier 4 was designed as a screening study to determine which combustion source categories emit CDDs and at what concentrations. The main focus was on releases to the ambient air; however, other samples, such as ash and scrubber water, were also obtained to determine if these compounds were released to other media. Because some combustion sources were known to emit a wide range of CDDs and CDFs, Tier 4 samples were analyzed for specific groups (homologs) of CDD and CDF compounds, as well as for 2,3,7,8-TCDD, the compound of most concern.

There are millions of combustion sources in the United States. Residential heating units burn oil, gas, coal and wood for heat. Larger commercial, institutional, and utility boilers burn fossil fuels to generate heat and electricity. Many industrial processes involve the combustion of fuels and other raw or waste materials to produce heat and/or recover products of marketable value. Other processes, such as incineration, use combustion to reduce the volume of unwanted waste products and to recover heat and

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003



	Statistical	Regional
Number of sites sampled	41	23
Number of sites contaminated* (•)	5	6
Number of sites not contaminated (o)	36	17

*More than 1 ppb in soil or detectable levels in other media.

Figure 2. Locations of Sampled Tier 3 Sites.

other resources from the waste products. Open fires, both accidental (e.g., structural and forest fires) and intentional (i.e., those set for forest management and agricultural burning), are other examples of combustion sources.

Assessment of CDD and CDF emissions from combustion sources has received limited study. Previous work included studies of emissions from hazardous waste incinerators, utility boilers, and municipal waste combustion. Even for those source categories that had previously been tested, there is considerable variation in the extent and quality of testing and the test methods employed.

Study Design. The large number of combustion source categories made it impractical to test each under Tier 4. A study plan was developed that identified those source categories that were believed to have the greatest potential for emitting CDDs to the atmosphere. Selection and prioritization of source categories for testing were based upon a review of CDD-related studies reported in the literature and engineering judgment. Information from this review suggested that the following conditions were most important for CDD formation:

- Presence of CDD in the materials being burned;
- Presence of CDD precursors in the materials being burned (e.g., chlorinated phenols, chlorinated benzenes); and
- Presence of chlorine, fuel, and combustion conditions conducive to CDD formation, including:
  - Relatively low combustion temperature (500-800°C);
  - Short residence time of fuel in the combustion zone (<1-2 seconds);
  - Lack of adequate oxygen (resulting in incomplete combustion);
  - Lack of adequate processing of fuels (e.g., burning of wet garbage); and
  - Lack of supplemental fuel to promote combustion efficiency.

Consistent sample collection procedures were used at each site selected. Sampling methodologies and procedures are described in three Tier 4 protocol documents. One document describes the ash sampling procedures; a second, the stack sampling procedures; and a third, the quality assurance measures and procedures (6). The stack testing method used at Tier 4 sampling sites is the state-of-the-art method, with minor modifications, proposed for use by a joint American Society of Mechanical Engineers (ASME) and EPA Work Group for municipal incinerators.

Results. All planned sampling efforts for Tier 4 have been completed. Approximately 350 CDD and CDF samples (20-25% of which were for internal quality assurance purposes) were collected. Thirteen sources were stack tested and 74 sites were tested under the ash sampling program. Collected samples were sent to the appropriate analytical laboratory in accordance with established procedures.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003

The results from each of the 13 sites stack tested under Tier 4 are presented in Table 3. Data presented in this table represent concentrations of emissions measured in the stack gases and are not representative of ground-level concentrations where people reside. In addition, the specific 2,3,7,8-tetra isomers for both CDD and CDF are presented in the respective tables.

These CDD/CDF stack concentrations have been normalized to a uniform stack oxygen concentration of 3 percent. This method removes the effect of dilution and is a more representative means of comparing what is being produced by the various combustion processes.

Table 4 lists the Tier 4 source in descending order of measured 2,3,7,8-TCDD concentration and provides total CCD and CDF concentrations.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003

Findings. This investigation included a review of information in the literature, as well as a sampling program designed to collect data for combustion source categories believed to have the greatest potential to emit CDDs and CDFs. Although all of the data have yet to be analyzed and fully interpreted, the following findings can be made:

- CDDs and CDFs are present in stack emissions from all sources tested in Tier 4 and most, but not all, of the combustion source categories reported in the literature.
- There is considerable variability in emission rates both among source categories and, in some instances, sources within the same source category. For example, the CDD emissions range from "nondetected" at seven coal-fired power plants tested (detection limit at less than 1 ng/dscm) to approximately 12,000 ng/dscm of total CDDs at a secondary copper smelter facility. Most of the combustion source categories fall within an intermediate range, generally two to three orders of magnitude less than the concentration at the secondary copper smelting facility.
- There is also considerable variability in the estimated risk to people residing in the vicinity of the sources tested. For most sources, risks to the most exposed individual were relatively low, i.e., in the range of 10^{-6} to 10^{-10}.
- CDD and CDF emissions from some sources have estimated risks to the most exposed individual of 10^{-5} or more. These sources include a secondary copper smelting facility, a sewage sludge incinerator, and some municipal incinerators. These findings are presently being considered by EPA with regard to whether CDD should be listed as a hazardous air pollutant under Section 112 of the Clean Air Act.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003

Table 3. Tier 4 CDD/CDF Stack Testing Results (6) (ng/dscm @ 3% O_2)[a]

Source	2,3,7,8-TCDD/ 2,3,7,8-TCDF	Chlorinated Dibenzo-p-dioxin/Dibenzofuran Homologs					
		Other Tetra-	Penta-	Hexa-	Hepta-	Octa-	Total[b] Tetra-Octa
Drum and Barrel Furnace	0.05/0.90	1.2/14	0.72/6.2	0.79/3.0	1.3/2.0	0.92/0.55	5.0/27
Industrial Carbon Regenerator	ND[c]/ND[c]	0.57/1.2	0.44/0.37	0.98/0.59	0.90/0.61	0.81/0.54	3.7/3.3
Industrial Waste Incinerator	4.5/21	77/570	100/610	150/650	230/470	61/66	630/2400
Kraft Paper Recovery Boilers							
Plant A	ND/0.02	ND/0.16	ND/0.06	0.06/0.07	0.18/0.16	0.49/0.13	0.73/0.59
Plant B	ND/0.01	ND/0.13	ND/ND	0.10/0.34	0.26/0.17	0.83/0.07	1.2 /0.71
Plant C	ND/0.01	0.13/0.46	0.15/0.46	0.39/0.59	0.88/0.50	1.4 /0.09	2.9 /2.1
Secondary Copper Smelter[d]	170/ 5,100	1,400/ 18,000	2,300/ 19,000	2,200/ 6,000	5,900/ 11,000	3,700/ 7,200	16,000/ 65,000
Sewage Sludge Incinerators							
Plant A	0.05/NR[e]	11/33	0.18/10	0.51/0.10	2.5 /0.50	5.3 /0.10	20/44
Plant B	ND/2.1	0.40/19	ND/4.8	ND/1.6	0.22/ND	0.98/0.07	1.6/28
Plant C	0.14/54	8.1 /150	1.1 /110	7.0 /32	21/60	15/45	53/450
Wire Reclamation Incinerator[d]	0.07/0.40	1.2 /29	2.2 /22	14/65	130/230	290/230	440/580
Wood-fired Boiler (Salt laden wood)	0.28/1.8	47/37	48/33	49/13	39/6.5	11/0.92	220/83
Wood Stoves	NR[e]/NR[e]	NR/NR	NR/NR	NR/NR	NR/NR	NR/NR	NR/NR

a = ng/dscm @ 3% O_2, nanograms per standard cubic meter of flue gas, normalized to 3-percent oxygen.
b = values across may not add up to totals due to rounding off.
c = ND, not detected, usually at less than 0.1 ng/dscm @3% O_2.
d = Estimated values; stack sampling results for this site do not meet analytical quality assurance objectives, but represent lower level estimates.
e = NR, not reported due to organic interference.

Table 4. Tier 4 and Other Sources Listed in Rank Order by 2,3,7,8-TCDD Concentrations (6) (ng/dscm @ 3% O_2)[a]

Source	2,3,7,8-TCDD	Total CDDs	Total CDFs
*Secondary Copper Smelter[b]	170	16,000	65,000
Municipal Waste Incinerator-Plant B	26	6,400	11,600
Municipal Waste Incinerator-Plant E	16	4,300	5,300
*Industrial Waste Incinerator	4.5	630	2,400
Hazardous Waste Incinerator	1.4	77	190
Municipal Waste Incinerator-Plant D	0.8	710	150
Municipal Waste Incinerator-Plant A	0.7	53	260
*Wood-fired Boiler	0.28	200	83
*Sewage Sludge Incinerator-Plant C	0.14	53	450
*Wire Reclamation Incinerator	0.07	440	580
*Sewage Sludge Incinerator-Plant A	0.05	20	44
*Drum and Barrel Furnace	0.05	5	27
Hospital Incinerator	ND[c]	330	735
Municipal Incinerator-Plant F	NR[d]	210	250
Municipal Incinerator-Plant C	NR	46	120
*Industrial Carbon Regenerator	ND	3.7	3.3
Municipal Carbon Regenerator	ND	3.3	4.8
*Kraft Paper Recovery Boiler-Plant C	ND	2.9	2.1
*Sewage Sludge Incinerator-Plant B	ND	1.6	28
*Kraft Paper Recovery Boiler-Plant B	ND	1.2	0.7
*Kraft Paper Recovery Boiler-Plant A	ND	0.7	0.6
(Co-fired Boiler)	ND	ND	ND
Coal-fired Utility Boilers (7 plants)	NR	ND	ND

[a]ng/dscm @ 3% O_2 = nanograms per standard cubic meter of flue gas normalized to 3 percent oxygen.
[b]Data reported for this site are "estimated minimum." The true value may be higher.
[c]ND = Not detected, generally at less than 1 ng/dscm @ 3% O_2.
[d]NR = Not reported.

- Considering the fact that Tier 4 focused on source categories and individual sources that were generally believed to have an above-average potential to emit CDD compounds, the results suggest that routine emissions of CDD and CDF compounds from many combustion source categories may not present a significant problem. However, since only a relatively small number of sources were tested, some sources that were not tested might also have CDD and CDF emissions that could result in elevated risks.

Future Work. Although the majority of the planned Tier 4 study has been completed, there are a number of continuing efforts that EPA plans to pursue with respect to CDD emissions from combustion sources. These include the following:

- A detailed technical report describing the Tier 4 program will be prepared once all of the data (e.g., ash data) has been analyzed and interpreted. Completion of the Tier 4 technical report is scheduled for late 1986.

American Chemical Society
Library
1155 16th St., N.W.
Washington, D.C. 20036

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003

- EPA is currently considering whether CDDs should be listed as a hazardous air pollutant category. A decision on whether to list CDD is expected by spring of 1987.
- EPA has a project underway to respond to the requirements of Section 102 of the Hazardous and Solid Waste Act of 1984 (HSWA) concerning CDD emissions from municipal incinerators. This effort is intended to identify design and operating guidelines to minimize CDD emissions.
- EPA will continue to coordinate with other environmental protection agencies (e.g., Environment Canada and the State of New York) in order to obtain the CDD/CDF test data that are currently being collected. It is expected that data for several municipal incinerator tests will be released by New York State later in 1986.
- EPA plans to continue its efforts to standardize and refine stack sampling and analytical procedures that will reflect improvements in the state-of-the-art. The recommended ASTM stack test methodologies for municipal incinerators are currently under EPA review and evaluation.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003

Tier 5, Use Sites

Tier 5 sites comprise areas where 2,4,5-TCP and pesticidal derivatives (including 2,4,5-T, Silvex, hexachlorophene, Erbon, Ronnel, and Isobac 20) were or are being used on a commercial basis. In this tier, a statistical sample was not possible due to the variety uses and conditions.

Tier 5 sampling was generally limited to those areas where the use of 2,4,5-T or Silvex has been documented, since information from the EPA Office of Pesticide Programs indicated that these two compounds have been more heavily used in specific areas and thus have a greater potential for causing significant human exposure to dioxin. The other compounds were of lesser interest due to: (a) low levels of active ingredient pesticide in the end-products; (b) use on very small areas; or (c) a wide diversity of uses at low levels of application. Lack of documentation on use also made it impractical to focus on these other compounds.

A total of 26 Tier 5 sites was sampled, including six forest sites, seven rice fields, two canals adjacent to sugarcane fields, one sugarcane field, three rights-of-way, three rangeland areas, one multiple use area, and three aquatic sites (used for recreation, fisheries, or multiple uses).

The environmental media to be sampled were determined by the EPA regional offices on a site-by-site basis. They included soils, stream sediments, fish tissue, vegetation, and animal tissue. All analyses were done at detection levels of approximately 1 ppt, because soil concentrations below 1 ppb can be of concern in certain types of areas, such as grazing lands.

Results. 2,3,7,8-TCDD has been detected at 15 of the 26 sites including two rights-of-way, one aquatic use site, two sugarcane fields, canals adjacent to one sugarcane field, four rice fields, three forest areas, one rangeland area, and one multiple use area.

Over 40 percent of the soil and sediment samples taken at contaminated sites had 2,3,7,8-TCDD present above the detection limit of approximately 1 ppt (5). Two sites had detectable levels in fish. At one of these, all fish samples were contaminated at levels up to 23 ppt in filets. 2,3,7,8-TCDD levels in soils at contaminated sites were between 0.6 and 564 ppt with 67 percent below 5 ppt; levels in sediments were between 0.7 and 200 ppt with 61 percent below 5 ppt; and levels in fish filets were between 8 and 23 ppt. No 2,3,7,8-TCDD was detected in animal tissue or vegetation samples collected from land used for grazing or raising crops; however, only a limited number of these samples was collected.

Findings. Since the source of 2,3,7,8-TCDD at the one severely contaminated Tier 5 site (Petenwell Flowage, Wisconsin) does not appear to be related to pesticide use, further national investigation of Tier 5 sites does not appear to be warranted. However, as described in greater detail under the Tier 7 discussion, further investigations of pulp and paper mills are being conducted.

Tier 6, "Other" Chemical Manufacturers

Tier 6 consists of organic chemical and pesticide manufacturing facilities where improper quality control within production processes could cause products or waste streams to become contaminated with 2,3,7,8-TCDD. Facilities producing any of sixty selected compounds were identified. The objective of the sampling for this tier was to determine the percentage of facilities with concentrations of 2,3,7,8-TCDD above 1 ppb in soil or at detectable levels in other environmental media (e.g., fish in nearby streams).

Findings. EPA estimates that 9 percent of the 66 facilities originally identified as Tier 6 sites are contaminated. None of the three sites was extensively contaminated with 2,3,7,8-TCDD (5). Based on these findings, further national investigation of Tier 6 sites for 2,3,7,8-TCDD does not appear to be warranted.

Tier 7, Background Sites

Tier 7 consists of sites that did not have previously known sources of 2,3,7,8-TCDD contamination. The Tier 7 investigation was intended to establish the prevalence of 2,3,7,8-TCDD in the environment and to provide a basis for comparison with results from the other tiers. The specific objectives of the sampling were to:

- Determine the percentage of sites in the EPA Urban and Rural Soil Networks that have detectable levels of 2,3,7,8-TCDD in soil at a detection limit of approximately 1 ppt.
- Determine the percentage of sites in the combined U.S. Geological Survey's (USGS) National Stream Quality Accounting Network and Benchwork Network that have detectable levels of 2,3,7,8-TCDD in fish tissue at a detection limit of approximately 1 ppt.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003

Findings. Of the statistically selected soil sites, 142 of 200 rural and 221 of 300 urban sites were sampled. The remaining 58 rural sites and 79 urban sites could not be sampled because of difficulty in locating the site (130 sites) or because permission to collect a sample was denied (7 sites). Of the 100 statistically selected fish sites, 90 were sampled. The remaining ten sites could not be sampled because of lack of water, fish, or success in catching fish at the time sampling was attempted.

Soil

- 2,3,7,8-TCDD was detected infrequently and at very low levels in background soil samples. 17 of 221 urban sites and 1 of 138 rural sites had detectable levels, with the highest level found being 11.2 ppt (5).

Fish

- 2,3,7,8-TCDD was detected more frequently in background fish samples. EPA estimates that 21 percent of the USGS national national monitoring network sites have detectable levels. The frequency of detection is greater (31 percent) at sites selected by EPA's regional offices, many of which are near industrial and urban areas.

- An even higher proportion (23 of 29) of Great Lakes' fish sampling sites had detectable levels, which is consistent with previous findings and is not surprising since the type and size of fish sampled in the Great Lakes and the long water retention times tend to increase the potential for bioaccumulation.

- 2,3,7,8-TCDD levels in filet samples could be a cause for concern at specific locations under certain consumption patterns; local exposure conditions need to be evaluated to determine a level of concern for those areas.

- Fish and shellfish from estuarine and coastal waters are rarely contaminated; two of the three sites where 2,3,7,8-TCDD was detected are in areas heavily influenced by industrial discharges.

- Certain types of pulp and paper mill discharges need to be further evaluated as possible sources of 2,3,7,8-TCDD. Both EPA and the industry are undertaking more detailed investigations.

Summary of Pertinent Regulations

As defined in the RCRA dioxin-listing rule (7), which became effective on July 15, 1985, the waste streams designated as acutely hazardous are:

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003

F020 - Wastes (except wastewater and spent carbon from HCl purification) from the production or manufacturing use (as a reactant, chemical intermediate, or component in a formulating process) of tri- or tetrachlorophenol, or of intermediates used to produce their pesticide derivatives. (This listing does not include wastes from the production of hexachlorophene from highly purified 2,4,5-TCP.)

F021 - Wastes (except wastewater and spent carbon from HCl purification) from the production or manufacturing use (as a reactant, chemical intermediate, or component in a formulating process) of pentachlorophenol, or of intermediates used to produce its derivatives.

F022 - Wastes (except wastewater and spent carbon from HCl purification) from the manufacturing use (as a reactant, chemical intermediate, or component in a formulating process) of tetra-, penta-, or hexachlorobenzenes under alkaline conditions.

F023 - Wastes (except wastewater and spent carbon from HCl purification) from the production of materials on equipment previously used for the production or manufacturing use (as a reactant, chemical intermediate, or component in a formulating process) of tri- and tetrachlorophenols. (This listing does not include wastes from equipment used only for the production or use of hexachlorophene from highly purified 2,4,5-TCP.)

F026 - Wastes (except wastewater and spent carbon from HCl purification) from the production of materials on equipment previously used for the manufacturing use (as a reactant, chemical intermediate, or component in a formulating process) of tetra-, penta-, or hexachlorobenzene under alkaline conditions.

F027 - Discarded unused formulations containing tri-, tetra-, or pentachlorophenol or discarded unused formulations containing compounds derived from these chlorophenols. (This listing does not include formulations containing hexachlorophene synthesized from prepurified 2,4,5-TCP as the sole component.)

F028 - Residues resulting from incineration or thermal treatment of soil contaminated with EPA hazardous waste codes F020, F021, F022, F023, F026, and F027.

Moreover, this rule replaced the regulation concerning the disposal of 2,3,7,8-TCDD-contaminated waste under the Toxic Substances Control Act (TSCA)--the so-called Vertac Rule--which was revoked on the same date. Unfortunately, the dioxin-listing rule currently does not cover thermally stressed PCBs, a source of CDDs and CDFs

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003

from transformer and capacitor fires (8) and in abandoned hazardous waste sites. The rule, however, may be modified to list this category in the future.

Land disposal of the listed wastes must be conducted pursuant to additional special standards implemented during the course of the permit proceeding. Since all of these wastes are specifically identified as candidates for being banned from land disposal in two years under the Hazardous and Solid Waste Amendment (HSWA) (see also RCRA-amended Section 3004 (e) and (h)(4)), EPA proposed an interim regulatory regime for their land disposal (9). This rule became final on November 8, 1986, and, as a result, wastes identified by the hazardous waste codes F020, F021, F022, F023, F026, F027, and F028 must be treated to a level below 1 ppb in the waste extract for each of the following specific categories of CDDs and CDFs:

TCDD--tetrachlorodibenzo-p-dioxins
TCDF--tetrachlorodibenzofurans
PeCDD--pentachlorodibenzo-p-dioxins
PeCDF--pentachlorodibenzofurans
HxCDD--hexachlorodibenzo-p-dioxins
HxCDF--hexachlorodibenzofurans

One ppb is the routinely achievable detection limit using method 8280 of SW-846: Test Methods for Evaluating Solid Waste Physical/ Chemical Methods (1982) (7, Appendix X). (In test method 8280, the proposed quantification level for 2,3,7,8-TCDD in water is 10 ppt. However, due to the interferences inherent in leachate samples and the variability of waste matrices, EPA considers that, generally, CDD wastes subject to this rule will have a detection limit of 1 ppb. It should be noted that because the treatment standard for CDDs is set at "no detection," it is important to calibrate to the levels specified in method 8280. An interlaboratory validation of the method has recently been conducted and reported (10).)

These listed wastes also must be treated below detection limits for 2,4,5-trichlorophenol, 2,4,6-trichlorophenol, 2,3,4,6-tetrachlorophenol, and pentachlorophenol. The detection limits for these constituents are 50, 50, 100, and 10 ppb, respectively, in the waste extracts using methods 3510/8270 identified in SW-846.

EPA also is granting the maximum two-year variance to the effective date of the land disposal restrictions (9) for dioxin-containing wastes because of a finding that there is a lack of capacity to treat and dispose of these wastes. Thus, the effective date of this final rule is November 8, 1988.

Literature Cited

1. U.S. Environmental Protection Agency (1983). Dioxin Strategy. Washington, DC, November 28.

2. Bellin, J.S. and Barnes, D.G. (1985). Procedures for estimating risks associated with exposures to mixtures of chlorinated dibenzodioxins and -dibenzofurans (CDDs

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003

and CDFs). Adopted by the Risk Assessment Forum, U.S. EPA, Washington, DC, November 21.

3. U.S. Environmental Protection Agency (1986). National Dioxin Strategy, Tier 1 and 2 Accomplishments. Draft Technical Support Document. Office of Solid Waste and Emergency Response, Washington, DC, January.

4. des Rosiers, P.E. (1985). Methodologies for materials contaminated with PCDDs and related compounds. Paper presented at the 5th International Symposium on Chlorinated Dioxins and Related Compounds, Bayreuth, Federal Republic of Germany, September 16-19.

5. U.S. Environmental Protection Agency (1986). The National Dioxin Study, Tiers 3, 5, 6, and 7. Draft Final Report. Office of Water Regulations and Standards, Washington, DC, April.

6. U.S. Environmental Protection Agency (1986). National Dioxin Study, Tier 4--Combustion Sources. Draft Project Summary Report. Office of Air Quality Planning and Standards, Research Triangle Park, NC, April.

7. U.S. Federal Register (1985), 50 (9), 1978-2006.

8. des Rosiers, P.E., Westfall, B., Campbell, B. and Lee, A. (1986). PCB fires: preliminary correlation of chlorobenzene and PCB contents of the fluid with PCDF and PCDD contents of soot. In: Proceedings: 1985 EPRI PCB Seminar, EPRI CS/EA/EL-4480, Project 2028, March, 7-26--7-39.

9. U.S. Federal Register (1986), 51 (9), 1602-1766.

10. Ballard, J.M., Vonnahme, T.L., Nunn, N.J., Youngman, D.R. and Billets, Stephen (1986). Performance of RCRA Method 8280 for the analysis of dibenzo-p-dioxins and dibenzofurans in hazardous waste samples. EPA/600/S4-86/021, July.

RECEIVED December 10, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch003

Chapter 4

Animal Toxicity Studies of 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin

Derivation of Lifetime Exposure Control Limit Recommendations for Humans

R. J. Kociba

Health and Environmental Sciences, Dow Chemical Company, Midland, MI 48674

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch004

Of the multiple toxicity studies performed with TCDD, those deemed most useful as the basis for derivation of human lifetime exposure control limit recommendations are the animal lifetime dose-response (carcinogenicity) studies, and the mechanistic studies wherein TCDD has been categorized as not being a mutagen nor possessing potential to interact directly with DNA. Multiple studies concur in reporting that the carcinogenic response noted in the animal studies with TCDD was reflective of a promoter mechanism rather than an initiator mechanism. While control limit recommendations for TCDD were originally derived by some regulatory agencies on a non-threshold assumption for extrapolation from the animal studies to humans, (assuming cancer initiation potential), more recently other regulatory agencies have utilized the newer data indicating a promoter (rather than initiator) mechanism as the valid basis for a threshold-based method of extrapolation from these animal studies to humans. Overall evaluation of all pertinent data indicates it is scientifically valid to derive the human lifetime control limit recommendations for TCDD through the use of a threshold-based process of extrapolation from the animal lifetime (carcinogenicity) studies.

Over the past fifteen years there have been extensive studies of the toxicologic properties of the 2,3,7,8-tetrachlorodibenzo-p-dioxin isomer (TCDD). Relatively speaking, less data are available for the other chlorinated dibenzo-p-dioxins and furans. These various toxicity studies have been the subject of several previous reviews (1, 2, 3, 4). The purpose of this present paper is to evaluate the basis upon which these animal toxicity studies have been utilized as the basis for extrapolation to man for the purpose of developing lifetime exposure limit control recommendations.

0097-6156/87/0338-0054$06.00/0
© 1987 American Chemical Society

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch004

Comparative Toxicity as Measured by Acute Lethality

The comparative data on the acute lethality of the chlorinated dibenzo-p-dioxins and furans in laboratory animals are included in Table I. Evaluation of these data indicates that TCDD is the most toxic of this series. For the guinea pig, the single dose oral LD50 value is reported to be 0.6-2.0 µg/kg B.W., while in the hamster the single dose oral LD50 value is reported to be 1157-5051 µg/kg B.W. The biologic factors that account for this large difference (up to 5000 fold) in species susceptibility in acute toxicity have not yet been defined. The other laboratory animal species (mouse, rat, monkey, rabbit and dog) in which TCDD has been tested have single dose oral LD50 values that are found to be somewhere between the most sensitive species (guinea pig) and the least sensitive species (hamster).

The question of likely sensitivity of man relative to the acute toxicity of TCDD in various laboratory animals has been addressed independently by Neal (17) and also by Tschirley (18). Both of these scientists have concluded from data on inadvertent human exposures that man appears to be less sensitive to TCDD than certain of the laboratory animals, such as the guinea pig. It is important to note that the most extensive animal studies of TCDD chronic toxicity/carcinogenicity used for extrapolation to man have been conducted with rats and mice, both of which represent species which are relatively more sensitive to TCDD toxicity. This fact indicates that the human exposure control limits derived from these chronic toxicity/carcinogenicity studies conducted in rats and mice supply an additional margin of safety inherent in the subsequent extrapolation process to man. Table I also lists the single dose oral LD50 values for the other chlorodibenzo-p-dioxins and furans that have been reported. Those tetra, penta, hexa and hepta isomers with the 2,3,7, and 8 positions chlorinated represent those isomers with greater toxicity as compared to the lesser chlorinated or fully chlorinated isomers that are considerably less toxic. This wide variation in acute toxicity of one isomer relative to another isomer is typified by the greater than six orders of magnitude differential between the LD50 values of 0.6-2 µg/kg for TCDD and the LD50 value of $>15\times10^6$ µg/kg for the 1,3,6,8-tetrachlorodibenzo-p-dioxin when tested in the very same species (guinea pig).

Comparative Biologic Activity as Measured by In Vitro Tests

Tables II and III are summary compilations of the relative biologic activities of chlorinated dibenzo-p-dioxins and furans, respectively, as measured by *in vitro* tests such as enzyme induction, epithelial keratinization or the "flat cell" effect in XB/3T3 cell cultures. For comparative purposes, the biologic activity of each isomer is expressed as a fraction relative to the biologic activity of TCDD in each type of test. Inspection of these data indicate that as a general rule the 2,3,7,8-substituted tetra, penta, hexa and hepta isomers have typically elicited the higher levels of biologic activity in these *in vitro* assays.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch004

Table I. Comparative Single Oral Dose LD50 Values For Chlorodibenzo-p-Dioxin And Chlorodibenzo-p-Furan Isomers

	Oral LD50 Values (μg/kg)							
Chlorodibenzodioxin	Guinea Pig	Mouse	Rat	Monkey	Hamster	Rabbit	Dog	Ref.
2,3,7,8-Tetra	0.6-2	114-284	22-45	70	1157-5051	115	>300,<3000	(5,6,7,8,9)
Unsub		>50,000	>1,000,000					(10,11)
2,3-Di			>1,000,000					(11)
2,7-Di		>2,000,000	>1,000,000					(5)
2,8-Di	>300,000	8,470,000	>5,000,000					(6,12)
1,3,7-Tri		>15,000,000	>5,000,000					(12)
2,3,7-Tri	29,444	>3,000	>1,000,000					(6,11)
1,2,3,4-Tetra			>1,000,000					(11)
1,3,6,8-Tetra	>15,000,000	>2,987,000	>10,000,000					(13)
1,2,3,7,8-Penta	3.1	337.5						(6)
1,2,4,7,8-Penta	1,125	>5,000						(6)
1,2,3,4,7,8-Hexa	72.5	825						(6)
1,2,3,6,7,8-Hexa	70-100	1250						(6)
1,2,3,7,8,9-Hexa	60-100	>1440						(6)
1,2,3,4,6,7,8-Hepta	>600							(6)
Octa		>4,000,000	>1,000,000					(5)
Chlorodibenzofuran								
2,8-Di		>15,000,000	>15,000,000					(12)
2,4,8-Tri		>15,000,000	>5,000,000					(12)
2,3,7,8-Tetra	5-10	>6000	>1000	1000				(14,15)
2,3,4,7,8-Penta	<10							(16)
2,3,4,6,7,8-Hexa	120							(16)

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch004

Table II. Comparative Biologic Activity (In Vitro) Of Chlorodibenzo-p-dioxins Relative To TCDD

Chlorodibenzo-p-dioxin	AHH Activity in Rat Hepatoma Cells	AHH Activity in Chick Embryo Liver	ALA Synthetase in Chick Embryo Liver	Keratinization of XB/3T3 Cells
2,3,7,8-Tetra	1/1	1/1	1/1	1/1
Unsub.	Inactive	Inactive	Inactive	Inactive
1-Chloro		Inactive	Inactive	
1,3-Di	Inactive			
1,6-Di	Inactive	Inactive		Inactive
2,3-Di	Inactive	Inactive	Inactive	Inactive
2,7-Di	Inactive	Inactive	Inactive	Inactive
2,8-Di	Inactive	Inactive	Inactive	
1,2,4-Tri		Inactive	Inactive	
2,3,7-Tri	1/920-1/3060	1/1666	Active	1/100
1,3,7,8-Tetra	1/57-1/242	1/12		1/100
1,2,3,8-Tetra	1/1666-1/5900			
1,2,3,4-Tetra	Inactive	Equiv.	Inactive	
1,3,6,8-Tetra	Inactive	Inactive	Inactive	Inactive
1,2,3,7,8-Penta	1/5-1/53			1/2
1,2,3,4,7-Penta	1/21-1/132	Active	Active	
1,2,4,7,8-Penta	Inactive			
1,2,3,6,7,9-Hexa	Inactive			
1,2,4,6,7,9-Hexa	Inactive	Inact./Equiv.	Inact./Equiv.	
1,2,3,4,7,8-Hexa	1/10-1/20	Active	Active	
1,2,3,7,8,9-Hexa	1/114-1/523	1/5		1/200
1,2,3,6,7,8-Hexa	1/71-1/947			
1,2,3,4,6,7,9-Hepta	1/10,200			
1,2,3,4,6,7,8-Hepta	1/282-1/367			
Octa (99.2%)	1/1666-1/4594			
Octa (>99%)	1/53,000	Inactive	Inactive	
	(Ref. 19, 20)	(Ref. 21, 22, 23)		(Ref. 24)

Biologic Activity expressed as fractions relative to TCDD (1/1).

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch004

Table III. Comparative Biologic Activity (In Vitro) Of Chlorodibenzofurans Relative To TCDD

Chlorodioxin/furan	AHH Activity in Rat Hepatoma Cells	AHH Activity in Chick Embryo Liver	Keratinization of XB/3T3 Cells	Flat Cell Effect in XBF/3T3 Cells
2,3,7,8-Tetra Dioxin	1/1	1/1	1/1	1/1
Chlorodibenzofuran				
Unsub.	Inactive	Inactive	Inactive	
2,8-Di	Inactive	Inactive		
2,4-Di		Inactive		
2,6-Di				<1/743
2,4,8-Tri			Inactive	
2,3,8-Tri	1/20,714			
2,4,6-Tri	Inactive			
1,4,6,8-Tetra	Inactive			
1,3,6,7-Tetra		Inactive		
2,3,6,8-Tetra	Inactive			
2,4,6,8-Tetra	Inactive			
2,3,7,8-Tetra	1/92	2/3	1/20	1/10
1,2,3,7,8-Penta		1/7		
1,3,4,7,8-Penta	1/1,928			
2,3,4,7,8-		7/10		
1,2,4,7,8-Penta	1/31,428			
2,3,4,6,7,8-Hexa				1/118
1,2,3,4,6,8,9-Hepta	1/24,286			
Octa				1/1400
	(Ref. 19)	(Ref. 21)	(Ref. 24)	(Ref. 25)

Biologic Activity expressed as fractions relative to TCDD (1/1).

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch004

Development and Interim Use of Toxicity Equivalent Factors

It is this type of comparative *in vitro* data on biologic activity as summarized in Tables II and III that has been used as the basis for deriving the concept of Toxicity Equivalent Factors (TEF) that has been proposed by certain regulatory agencies in lieu of generating chronic toxicity data on each isomer of concern. Table IV depicts the TEFs currently being proposed by US EPA for the chlorodibenzo-p-dioxins and furans (26). Although the concept of using the TEF (derived from short term or in vitro tests) may seem to be a readily available and attractive interim procedure, two recent studies have both reported a lack of correlation between the TEF predicted toxicity and the actual toxicity observed in subchronic animal studies. The first of these two studies has been reported by Doyle and Friess (27) and is summarized in Table V. In these studies the subchronic animal toxicity actually observed was considerably less than what had been predicted by the enzyme induction assays. Suter-Hofman (28) also reported a lack of correlation between the toxicity predicted by the use of TEF when compared to the actual toxicity observed in a subchronic toxicity test wherein animals were given a mixture of dioxin and furan isomers. In this study, a toluene extract of municipal incineration particulate emissions was fed to rats. Analysis indicated the extract to contain 2,3,7,8-TCDD as well as additional chlorinated dioxins and furans at concentrations that were calculated to be supplying a dose level of 65 ng of 2,3,7,8-TCDD/kg BW/day or a composite dose level representing all the chlorinated dioxins and furans of 2000 ng of 2,3,7,8-TCDD TEF/kg BW/day. Using previous rat toxicity studies with 2,3,7,8-TCDD as the reference point, the extract caused only a slight degree of toxicity of the magnitude predicted for a dose level of 65 ng of 2,3,7,8-TCDD/kg BW/day. The severe toxicity and lethality predicted by the use of the TEF scheme to be associated with a composite dose level of 2000 ng of 2,3,7,8-TCDD TEF/kg BW/day was not observed in the rats. Thus, it appears as if the 2,3,7,8-TCDD TEF scheme (based on *in vitro* assays) for total chlorinated dioxins and furans substantially overpredicts the actual toxicity observed when *in vivo* studies of combinations of multiple dioxins and furans are conducted.

Other Toxicity Data on TCDD and Related Compounds

The extensive toxicologic data available on TCDD and other dioxin and furan isomers from animal tests for rabbit ear chloracnegenicity, teratogenicity/fetotoxicity, reproductive toxicity, mutagenicity, clastogenicity, and carcinogenicity have been compiled and reviewed in previous publications (1, 2, 3, 4). Because of space limitations these data cannot be completely reviewed here, and the previous reviews should be consulted for specific details. Overall, the concensus is that the qualitative and quantitative aspects of these various toxicologic endpoints have been adequately defined for TCDD.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch004

Table IV. Toxicity Equivalent Factors (TEF) Proposed By U.S. EPA For Isomers Of Most Interest

Isomer	TEF[a]	Isomer	TEF
2,3,7,8-TCDD	1	2,3,7,8-TCDF	0.1
1,2,3,7,8-PeCDD	0.5	1,2,3,7,8-PeCDF	0.1
		2,3,4,7,8-PeCDF	0.1
1,2,3,4,7,8-HxCDD	0.04	1,2,3,4,7,8-HxCDF	0.01
1,2,3,7,8,9-HxCDD	0.04	1,2,3,7,8,9-HxCDF	0.01
1,2,3,6,7,8-HxCDD	0.04	1,2,3,6,7,8-HxCDF	0.01
		2,3,4,6,7,8-HxCDF	0.01
1,2,3,4,6,7,8-HpCDD	0.001	1,2,3,4,6,7,8-HpCDF	0.001
		1,2,3,4,7,8,9-HpCDF	0.001

In each homologous group the relative toxicity for the isomers not listed above is 1/100 of the value listed above.

[a]TEF = Toxic Equivalent Factor = relative toxicity assigned.

(Ref. 26)

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch004

Table V. Lack Of Predictive Correlation Between AHH Enzyme Induction And 90 Day Feeding Studies In Rats

Chlorodioxin/Furan	Rat Liver AHH Enzyme Induction Activity Relative to TCDD	Comparative Results of 90-Day Feeding Studies in Rats
2,3,7,8-TCDD	1/1	30 ppb-Expected toxic syndrome, with 25-50% mortality, wt. loss, adverse effects on organ wts, blood and serum parameters
2,3,7,8-TCDF	1/2	60 ppb-Only minor effects-Increased relative liver wts in males
2,7-DiCDF	1/2	60 ppb-No significant effects
2,8-DiCDF	1/5	60 ppb-No significant effects
OctaCDF	1/5	60 ppb-No significant effects
1,2,4,7,8-PentaCDF	Inactive	60 ppb-No significant effects
1,2,3,4,6,7,9-HeptaCDF	Inactive	60 ppb-No significant effects

Results of 90 day feeding studies indicate these chlorodibenzo furans were considerably less toxic than might be predicted from their AHH enzyme induction activity. (Ref. 27).

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch004

Use of Animal Toxicity Data for Deriving Exposure Control Limit Recommendations For Humans

For purposes of this review the emphasis will be on those toxicity studies upon which long term or chronic lifetime exposure control limit recommendations have been derived. Thus, the animal data on reproductive toxicity (29, 1, 4), chronic toxicity and carcinogenicity (30, 2, 3), mutagenicity (2, 3, 31) and related mechanistic data (2, 3) are the most pertinent to this discussion. TCDD, as well as various other chlorinated dioxins and furans have been studied extensively in various assays designed to detect mutagenic and clastogenic types of activity. The preponderance of these multiple studies indicates that TCDD (and the other dioxins and furans) does not possess any significant mutagenic/clastogenic potential (2, 3, 4, 31). Other studies have reported a lack of covalent binding of TCDD to DNA or RNA, with maximal binding potential 4-6 orders of magnitude less than most genotoxic carcinogens (32). Likewise, TCDD did not stimulate unscheduled DNA synthesis when tested in rat hepatocytes (33) or in a human cell line (34). The various studies with TCDD in regard to tumor initiation, promotion and cocarcinogenesis that have been reviewed previously (2, 3, 4) indicate that TCDD is more correctly categorized as a tumor promoter rather than an initiator. Of the four chronic animal carcinogenesis studies that have been conducted with TCDD, the one that has been used by essentially all regulatory agencies for derivation of lifetime exposure control limit recommendations is the study by Kociba et al. (30). The pertinent results of that study are summarized in Table VI in a manner that correlates dose levels, observed response (carcinogenicity, toxicity, no-adverse effect level) and terminal tissue levels of TCDD. A lifetime daily dose level of 0.1 μg/kg/day of TCDD caused both toxicity and carcinogenicity, whereas a lifetime dose level of 0.01 μg/kg/day of TCDD caused some toxicity but no definitive carcinogenic response. A lifetime dose level of 0.001 μg/kg/day of TCDD caused no toxicity or carcinogenicity and was defined as the no-observed-adverse-effect-level (NOAEL) in this lifetime study in rats. The NOAEL of 0.001 μg/kg/day defined in this lifetime study (30) is the same NOAEL defined in a multigeneration reproduction study in rats (29). Using this lifetime study of TCDD in rats (30) the U.S. Environmental Protection Agency (EPA), U.S. Food and Drug Administration (FDA), U. S. Center for Disease Control (CDC), Ontario Ministry of the Environment (OMOE), Dutch Government, the Federal Republic of Germany and the Swiss Institute of Toxicology have all derived carcinogenicity based lifetime exposure control limit recommendations. Table VII is a compilation of these carcinogenicity-based control limit recommendations issued by these groups. The U.S. agencies have followed their previous policy decisions to regulate all suspect carcinogens on a non-threshold basis, and have utilized a multistage linearized model to calculate the upper limit lifetime cancer risk estimates. Data summarized in Table VII indicates that this modelling procedure projects that a "virtually safe dose" level of exposure (one additional cancer in one million lifetimes) equates with lifetime exposure to 0.0064, 0.0572 or 0.0276 pg/kg BW/day, respectively, depending on the assumptions

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch004

Table VI. Correlation Of Dosage, Response And Tissue Levels Of TCDD At Termination Of Two-Year Toxicity Study In Rats Used As Basis For Derivation Of Cancer-Based Lifetime Exposure Control Limit Recommendations

Dose Levels of TCDD		Response		Terminal TCDD Content ppt	
µg/kg/day	ppt in Diet	Carcinogenicity	Other Toxicity	Fat	Liver
0.1	2200	Yes	Yes	8100	24000
0.01	210	No*	Yes	1700	5100
0.001	22	No	No	540	540

*Maximal response of hyperplastic nodules of liver in female rats.
(Ref. 30)

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch004

Table VII. Procedures In Use For Extrapolation To Man From Chronic Animal Studies With TCDD

Agency/Group	Source of Dose/Response Data	Mechanistic Basis for Extrapolation to Man	Results of Extrapolation Process
U.S. EPA (Ref. 35)	2 year rat study Kociba et al. (Ref. 30)	Non Threshold, Linearized, Multi-stage model (dose/surface area)	V.S.D. for upper-limit cancer risk of 10^{-6}=0.0064 pg/kg/day
U.S. FDA (Ref. 36)	Same	Non Threshold, Linearized, Multi-stage model (dose/body weight)	V.S.D. for upper-limit cancer risk of 10^{-6}=0.0572 pg/kg/day
U.S. CDC (Ref. 37)	Same	Non Threshold, Linearized, Multi-stage Model (liver conc.)	V.S.D. for upper-limit cancer risk of 10^{-6}=0.0276 pg/kg/day
Ontario (Ref. 38)	Same	Threshold, safety factor applied to NOEL	Maximal allowable daily intake=10 pg/kg/day
W. Germany (Ref. 39)	Same	Threshold, safety factor applied to NOEL	Max. daily intake= 1-10 pg/kg/day
Netherlands (Ref. 40)	Same	Threshold, safety factor applied to NOEL	Allowable daily intake= 4 pg/kg/day
Swiss Institute of Toxicology (Ref. 41)	Same	Threshold, safety factor applied to NOEL	Tolerable daily intake= 10 pg/kg/day max.

inherent in each of the extrapolation procedures used by these 3 U.S. agencies.

In contrast to the U.S. agencies cited above, more recent risk assessments conducted by Ontario, West Germany, Dutch and Swiss agencies have used a threshold basis for extrapolation from the same chronic animal study (30). This threshold based extrapolation has been based on use of more recent mechanistic data indicating that TCDD is more correctly categorized as a cancer promoter with a threshold for its promoter activity rather than a cancer initiator. Using this threshold based extrapolation, the four non-U.S. regulatory agencies or groups listed in Table VII have established a lifetime acceptable daily intake (or maximum daily intake or allowable daily intake or tolerable daily intake) for TCDD (or its toxic equivalents) in the range of 1-10 pg/kg BW/day.

Review of all pertinent data indicates that, based on the demonstration that TCDD acts as a promoter rather than an initiator, a threshold-based safety factor approach is considered the most scientifically appropriate to be used with TCDD when setting an adequate margin of safety to protect the long-term health of the general population. In this regard, the threshold-based approach as utilized by the four non-U.S. agencies is considered the most valid basis for the animal to man extrapolation. In the report issued by the Ontario MOE (38) their expert panel concluded that TCDD produces tumors in rodents by an indirect mechanism, with a threshold level of dosage (0.001 μg/kg/day, or 1 ng/kg/day) below which an increased rate of tumor production due to exposure to TCDD would be unlikely. The NOAEL as defined in the chronic animal studies was used as an indication of where this threshold lies. To this NOAEL, application of a safety factor of 1/100 was considered appropriate in calculating the maximum allowable daily intake for lifetime exposure of humans. This 1/100 safety factor applied to the NOAEL incorporates a number of uncertainties in extrapolating from animal data to humans, particularly an allowance for those segments of the human population that may possibly have an increased level of sensitivity. Application of this 1/100 safety factor to the NOAEL (1 ng/kg/day or 1000 pg/kg/day) defined in the lifetime rat study (30) that is considered most definitive by essentially all of the regulatory agencies was the basis for the Ontario MOE expert panel to recommend a Maximum Daily Intake for 2,3,7,8-TCDD (or its toxic equivalent of other chlorinated dioxins and furans) of 10 pg/kg B.W./day for humans (38). This more recent approach used by these four non-U.S. groups demonstrate the manner in which carcinogenicity - based lifetime exposure control limit recommendations for humans can be realistically derived from the data available from the animal cancer bioassays when interpreted in concert with the data available regarding the likely mechanism of action by which the carcinogenic response occurred in the animal bioassays.

Literature Cited

1. Kociba, R. J.; Schwetz, B. A. Assoc. Fd. Drug Offic. Qrtrly, Bull. 1982, 46, 168-188.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch004

2. Kociba, R. J. In "Public Health Risks of the Dioxins"; Lowrance, W. Ed.; Wm. Kaufmann: Los Altos, 1984, p. 77.
3. Kociba, R. J. In "Biological Mechanisms of Dioxin Action", Poland, A. and Kimbrough, R. D. Ed.; Banbury Center, Cold Spring Harbor Laboratory: New York, 1984; p. 73.
4. Kociba, R. J.; Cabey, O. Chemosphere 1985, 14, 649-660.
5. Schwetz, B. A.; Norris, J. M.; Sparschu, G. L.; Rowe, V. K.; Gehring, P. J.; Emerson, J. L.; Gergib, C. G. Environ. Hlth. Perspect. 1973, 5, 87-99.
6. McConnell, E. E.; Moore, J. A.; Haseman, J.; Harris, M. W. Toxicol. Appl. Pharmacol. 1978, 44, 335-356.
7. McConnell, E. E.; Moore, J. A.; Dalgard, D. W. Toxicol. Appl. Pharmacol. 1978, 43, 175-187.
8. Henck, J. W.; New, M. A.; Kociba, R. J.; Rao, K. S. Toxicol. Appl. Pharmacol. 1981, 59, 405-407.
9. Olson, J. R.; Holscher, M. A.; Neal, R. A. Toxicol. Appl. Pharmacol. 1980, 55, 67-78.
10. Courtney, K. D. Bull. Environ. Contam. Toxicol. 1976, 16, 674-681.
11. Saint-Ruff, S. In "Dioxin: Toxicological and Chemical Aspects"; Cattabeni, F., Cavallaro, A., Galli, G., Ed.; Spectrum Public: New York, 1978, Chapter 15, p. 77.
12. Bernstein, M. Summary of Toxicity Data, Ciba Geigy Corp., New Jersey, 1981.
13. Kawamura, K.; Sato, R.; Koshima, M. Oyo Yakuri 1983, 25, 703-711.
14. "Toxicity of 2,3,7,8-Tetrachlorodibenzofuran-Preliminary Results" U. S. EPA Report 56016-75-004, 1976.
15. Moore, J. A.; McConnell, E. E.; Dalgard, D. W.; Harris, M. W. Ann. N.Y. Acad. Sci. 1979, 320, 151-163.
16. McKinney, J.; McConnell, E. E. In "Chlorinated Dioxins and Related Compounds"; Hutzinger, O., Frei, R. W., Merian, E., Pocchiara, F., Ed.: Pergamon Press: New York, 1981; p. 367-381.
17. Neal, R. Interim Report of the Missouri Dioxin Task Force, 1983, p. 40-41.
18. Tschirley, F. H. Scientific American 1986, 254, 29-35.
19. Bradlaw, J.; Casterline, J. J. Assoc. Off. Analyt. Chem. 1979, 62, 904-916.
20. Bradlaw, J.; Garthoff, L.; Hurley, H.; Firestone, D. Fd. Cosmet. Toxicol. 1980, 18, 627-635.
21. Poland, A.; Glover, E. Mol. Pharmacol. 1973, 9, 736-747.
22. Poland, A.; Glover, E. J. Biol. Chem. 1976, 251, 4936-4946.
23. Poland, A.; Kende, A. Fed. Proc. 1976, 35, 2404-2411.
24. Knutson, J.; Poland, A. Cell 1980, 22, 27-36.
25. Gierthy, J. F.; Crane, D. Fund. Appl. Toxicol. 1985, 5, 754-759.
26. "Procedures of Estimating Risks Associated with Exposure to Mixtures of Chlorinated Dibenzodioxins and Dibenzofurans", U.S. Environmental Protection Agency, 1985.
27. Doyle, E. A.; Fries, G. F. 5th Intern. Symp. on Chlorinated Dioxins, 1985, Abst. 116.
28. Suter-Hofman, M. 5th Intern. Symp. on Chlorinated Dioxins, 1985, Abst. 115.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch004

29. Murray, F. J.; Smith, F. A.; Nitschke, K. D.; Humiston, C. G.; Kociba, R. J.; Schwetz, B. A. Toxicol. Appl. Pharmacol. 1979, 50, 241-251.
30. Kociba, R. J.; Keyes, D. G.; Beyer, J. E.; Carreon, R. M.; Wade, C. E.; Dittenber, D. A.; Kalnins, R.; Frauson, L. F.; Park, C. N.; Barnard, S. D.; Hummel, R.; Humiston, C. G. Toxicol. Appl. Pharmacol. 1978, 46, 279-303.
31. Wasson, J. S.; Huff, J. E.; Loprieno, N. Mut. Res. 1978, 47, 141-160.
32. Poland, A.; Glover, E. Cancer Res. 1979, 39, 3341-3344.
33. Althaus, F. R.; Lawrence, S. D.; Sattler, G. L.; Longfellow, D. G.; Pitot, H. C. Cancer Res. 1982. 42, 3010-3015.
34. Loprieno, L.; Sbrana, I.; Rusclano, D.; Lasciafari; Lari, T. In "Chlorinated Dioxins and Related Compounds"; Hutzinger, O., Ed.: Pergamon Press: New York, 1983, Vol. 5.
35. Hiremath, C. B.; Bayliss, D. L.; Bayard, S. 5th Intern. Symp. on Chlorinated Dioxins, 1985, Abst. 131.
36. "Statement by Sanford A. Miller Before the Congressional Subcommittee on Natural Resources, Agriculture Research and Environment", U.S. Food and Drug Administration, 1983.
37. Kimbrough, R. D.; Falk, H.; Stehr, P.; Fries, G. J. Toxicol. Environ. Hlth. 1984, 14, 47-93.
38. "Scientific Criteria Document for Standard Development No. 4-84-Polychlorinated Dibenzo-p-dioxins (PCDDs) and Polychlorinated Dibenzofurans (PCDFs)", Ontario Ministry of the Environment, 1985.
39. Appel, K. E.; Beck, H.; Hildebrandt, A. G.; Lingk, W. 5th Intern. Symp. on Chlorinated Dioxins, 1985, Abst. 132.
40. Evaluation of the carcinogenicity and mutagenicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD); classification and no-effect level" Report DOC/LCM 300/292, Dutch State Institute of National Health, 1982.
41. Schlatter, C.; Poiger, H. Assessment of Contamination with 2,3,7,8-TCDD, Institute for Toxicology, University of Zurich, 1985.

RECEIVED November 25, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch004

Chapter 5

Effects of Chlorinated Dibenzodioxins

Renate D. Kimbrough and Vernon N. Houk

Center for Environmental Health, Centers for Disease Control, Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA 30333

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch005

This paper is a review of the reported effects of 2,3,7,8-tetrachlorodibenzodioxin (TCDD) on the health of workers in chemical plants and on the general population. Virtually no information exists on the human health effects of other congeners of polychlorinated dibenzodioxins (PCDDs). Most exposures associated with acute health effects have been occupational, particularly those occurring when reaction vessels have exploded during the production of 2,4,5-trichloro-phenol. Symptoms of 2,3,7,8-TCDD exposure are chloracne, severe fatigue, nervousness, and decreased libido. In addition, workers may complain of weakness, loss of appetite, sleep disturbances, and a sensory neuropathy. In two factories, porphyria cutanea tarda was reported in workers. It is not clear, however, whether this illness was caused by the TCDD and/or by hexachlorobenzene and other chemicals, which were also present in the plants. Follow-up studies of highly exposed workers indicated that chloracne was the most persistent health effect. Upon cessation of exposure, many of the other abnormalities either decreased in severity or subsided completely. In a few isolated incidences, members of the general population have been exposed to significant levels of TCDD. In one such episode in Seveso, Italy, the systemic illness was mild and apparently short-lived. In another episode, a child had a hemorrhagic cystitis, and a few others had chloracne. Both of these symptoms subsided. No convincing chronic health effects have been reported. In case-control studies, an association has been made between soft-tissue sarcoma and exposure to phenoxy herbicides that contained TCDD. Subsequent studies, however, have cast some doubt on these findings, and further studies are needed. There are 75 isomers of chlorinated dibenzodioxins (PCDDs). They vary greatly in toxic ity. The most toxic isomer is the 2,3,7,8-tetrachlorodibenzodioxin (2,3,7,8-TCDD). Other isomers, in which at least three of the 2,3,7,8 positions are occupied by halogens, are also quite active biologically. Most of what we know about the toxicity of these chemicals was developed from animal studies, and most studies conducted were done with the 2,3,7,8-TCDD. Different species respond quite differently to the toxic effects of 2,3,7,8-TCDD. The hamster,

This chapter not subject to U.S. copyright.
Published 1987, American Chemical Society

for instance, appears to be 1,000 times less sensitive to the toxic effects of 2,3,7,8 TCDD than is the guinea pig. The sensitivity of humans to 2,3,7,8-TCDD is still unknown. From animal studies we have learned that 2,3,7,8-TCDD causes liver toxicity, affects the skin, depresses the cell-mediated immune response, affects reproduction and cell differentiation, and produces cancers in rats and mice (1). But the target organs in different animal species vary and, except for the skin and, perhaps, the liver, it is not clear what other health effects these chemicals may have in humans. It is also unclear what health effects the other PCDDs might have singly or in combination.

Sources

PCDDs are accidentally formed during the production of chlorinated phenols and related products (2-5). They may also be formed during the burning process in incinerators and through other forms of combustion, such as motor vehicles. The 2,3,7,8-TCDD is usually a minor component of these combustion products. They have been identified in urban dust, in gasoline motor oil, and in diesel motor oil (Ballschmiter, K.; Buchert, H.; Niemczy, K. R.; Munder, A.; Swerrev, M. Chemosphere in press 1986.) Thus, these chemicals are ubiquitous in our environment in very low concentrations and have been identified in fish, lake sediments, human adipose tissue, and milk (6-10). In addition, waste from the production of chlorinated phenols, if poorly controlled, may lead to heavier contamination in local areas (11).

Human health effects

Exposure to PCDDs may occur in the occupational as well as the general environment. The general population is usually exposed to lower levels than workers. For this reason, workers are the ones primarily examined for acute and chronic health effects, unless subgroups of the general population for some reason have higher exposures.(12).

For most of the PCDDs no information on human health effects is available except that during the production of pentachlorophenol workers have developed a skin disease, chloracne (13) and complaints such as neuralgic pain in the lower extremities, and symptoms such as persistent bronchitis, and eye irritation (14). The bronchitis seems to be more prevalent in workers exposed to technical pentachlorophenol than in those exposed to the other chlorinated phenols.

Humans with a history of exposure to 2,3,7,8-TCDD have been studied most often. Therefore, I will devote most of the rest of this paper to a review of these studies. Although exposure to other PCDDs may be as prevalent or more prevalent in the general population, we have no information on the human health effects of the other PCDDs.

Workers can be exposed to PCDDs during the manufacture of an industrial product, while mixing ingredients for a commercial product, or when packaging the commercial product. The doses that these different populations receive vary widely. Occasionally,

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch005

maintenance workers and cleanup crews may receive high exposures to such chemicals, which could be avoided through the use of appropriate gear and the practice of good personal hygiene.

Another hazard is peculiar to 2,3,7,8 TCDD. During the production of 2,4,5-trichlorophenol, an exothermic reaction can occur, causing the reaction vessel to explode. This has happened at several production facilities. In almost all of these incidents workers were heavily exposed to 2,3,7,8 TCDD during the cleanup (15-20). Chloracne has developed in the workers, and, in some instances, acute systemic disease.
(Chloracne is a skin disease which consists of small skin-colored cysts with a central opening and of blackheads).

Chloracne is produced either by direct contact with the chemical on the skin or as an expression of a systemic disease. Another group of chemicals, the chlorinated biphenyls and the chlorinated dibenzofurans, also produce chloracne. Two outbreaks of poisoning by these chemicals occurred in the general population in Japan and Taiwan. In both instances, the offending chemicals were ingested and generalized chloracne developed in the patients.

The clinical features of chloracne, regardless of the type of chemical causing it, have been most consistent. The most distinctive cutaneous lesion is a skin-colored cyst that measures from 1 to 10 mm in diameter, with a central opening.

The other dominant lesion is the comedo. Usually these lesions start over the maxillary bone, then involve the entire face and the neck. Lesions may also be present on the back, arms, legs, and other areas of the body. Large pustules may form because of secondary infection.

In areas exposed to sunlight, a photosensitivity-type reaction may occur. Conjunctivitis, with swelling of the eyelids and other facial skin, may precede the chloracne.

Once chloracne has developed, it may be active for many years. Often, secondary infections occur, leaving deep-pitted, permanent scars.

In addition to chloracne, acute or chronic health effects, or both, may occur. Acute effects are noticed soon after heavy exposure to TCDD, as after the explosions (21). In such cases, workers usually become ill within 1 week. If workers are continually exposed to lower levels of TCDD, however, signs of illness may not occur for several months. Under such circumstances, the overriding sign is the chloracne. Although in Nitro, West Virginia exposure and occupational illness occurred as early as 1947, it was not until 1957 that TCDD was identified as the culprit (19).

Dermatologists have conducted most investigations of acute poisoning episodes, and their reports primarily focus on the skin lesions. Whether, occasionally, illness without chloracne may occur in exposed individuals is, at present, a matter of debate. In these outbreaks, some workers had severe chloracne without any systemic effects. On the other hand, ailments involving the skin, the eyes, the liver, the nervous system, and, occasionally, the heart were observed in other workers. The eye-related problems included conjunctivitis (inflammation of the conjunctiva) and blepharitis (inflammation of the eyelids).

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch005

In addition to the chloracne, hyperpigmentation and hirsutism (increased hair growth) were also reported.

The liver is most consistently affected in severe acute 2,3,7,8-TCDD poisoning. Goldmann et al. (15) described morphological changes in a liver biopsy from an affected worker as mild subacute hepatitis with focal areas of hyaline. The hepatocytes were enlarged, with moderately sized lipid vacuoles in their cytoplasm. A gray pigment that did not stain positive for iron was noted.

Many patients also had a sensory neuropathy (functional and/or pathological changes in the nervous system). In addition to the neuropathy, neurasthenia (functional nervous debility) has been associated with acute 2,3,7,8-TCDD poisoning. Complaints related to neurasthenia have been sleep disturbances, decreased libido, impotence, lack of drive, and mood changes. Unfortunately, these types of complaints cannot be objectively substantiated. They are often the types of complaints associated with aging or with depression and thus they are difficult to evaluate. In many instances, the skin lesions were quite disfiguring, and this may have caused a depression in some patients.

Paresthesias (abnormal sensations such as burning prickling), headache, muscle pain, and leg weakness have been described in workers acutely exposed to 2,3,7,8-TCDD. Loss of appetite and diarrhea were occasionally observed. Most of the other organs did not seem to be affected. In contrast, May (16), reported a poisoning outbreak in which workers suffered severe chloracne but had no other obvious systemic illnesses.

In the only poisoning outbreak in which humans (three chemists) were exposed solely to 2,3,7,8-TCDD, chloracne developed in two patients (22), and they had hyperpigmentation, increasing fatigue, and a tendency to have headaches. One patient complained that his skin was excessively oily. One of these patients lost about 6.5 kg of weight and reported excessive fatigue and loss of vigor. He also had excessive hair growth (hirsutism). About 1 year later, serum cholesterol levels for all three were raised, but clinical laboratory tests showed no other abnormalities. These chemists had been synthesizing 2,3,7,8-TCDD, an activity that poses the greatest hazard of exposure for chemists.

Patients who showed an increase in total serum lipids have also been described by Jirasek et al. (23) and Pazderova-Vejlupkova et al. (24). Many of these patients had abnormal results for glucose tolerance tests, hepatic lesions, increased alpha-1 and gamma globulin in plasma, and decreased plasma albumin. Hypercholesteremia and polyneuropathy were quite prevalent.

In two factories, in addition to chloracne, porphria cutanea tarda (a disturbance of porphyrin metabolism characterized by chronic skin lesions ranging from slight skin fragility to severe chronic scarring, by enlarged livers and by excessive urinary excretion of uroporphyrin and coproporphyrins. Uroporphyrin and coproporphyrin are iron-free cyclic tetrapyrrole derivatives) developed in the workers, (23,25). The possibility of simultaneous exposure to hexachlorobenzene, however, cannot be excluded. Hexachlorobenzene may be a contaminant in the production of

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch005

pentachlorophenol. In the episode described by Jirasek et al. (23), pentachlorophenol was also produced, and four patients with severe porphyria cutanea tarda also had hirsutism but did not have chloracne, this was similar to the reaction of patients who had been poisoned by hexachlorobenzene (26,27). In many of these factories, follow-up studies were conducted (15,17,24,28-31).

Pazderova-Vejlupkova et al. (24) studied 55 exposed workers 10 years after their illnesses began. Most workers' health had improved. Their serum lipid levels did not significantly differ from those of the controls. Their serum alpha and gamma globulins had normalized, but elevated mean values of total blood protein were still present. Increased concentrations of porphryins were no longer present in urine, skin mainfestations normally seen with porphyria cutanea tarda were rare, and results of liver function tests had improved. In the liver biopsies, even for the most severe forms of poisoning, only mild steatosis or periportal fibrosis were noted.

In 1947 at a Nitro, West Virginia, plant an explosion occurred during the manufacture of 2,4,5-trichlorophenol. The accident was reevaluated in 1984 (30). Chloracne still persisted in 55.7% of the workers. Other investigators have also noted this extreme persistence of chloracne (24,28). Regarding systemic health effects, not much was found. Hypertension, angina, coronary artery disease, skin cancer, and cancers of all sites were not significantly associated with exposure. Among those who had been exposed, 59.1% had actinic elastosis (degeneration of elastic tissue in the skin caused by those rays of light beyond the violet end of the spectrum that produce chemical effects), in contrast to 30.1% among the controls. The laboratory findings were essentially within the normal range. After adjustment for age and smoking, the results of nerve-conduction tests for the controls and the exposed groups did not differ. The pulmonary function values among those who were exposed and who currently smoked, however, were lower than values for those who were not exposed and who did not smoke.

Moses et al. (32) studied a portion of the same cohort that had been reexamined by Suskind and Hertzberg (30). In 1979, 226 workers were examined and chloracne was still present in 52%. The chloracne in many of these workers had persisted for 26 years; in 29 workers, it had persisted for 30 years. These workers also showed elevated gamma glutamyl transpeptidase. The mean gamma glutamyl transpeptidase was higher in workers with chloracne than in those without. This survey revealed no other findings of note. The difference in the results between the follow-up by Moses et al. and the one by Suskind and Hertzberg can be explained, in part, by the difference in the composition of the cohorts.

May (29), conducted a similar study 10 years after an explosion in a factory in which chloracne developed in 79 workers, and found that only half of the affected workers still had chloracne. No other adverse health effects were noted that could be related to the previous exposure.

The mortality data for these different groups did not indicate any obvious trends. However, Thiess et al. (18) reexamined 74 persons whom Goldmann (15) had previously studied. These workers

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch005

had been exposed to TCDD during an uncontrolled reaction at a trichlorophenol facility several decades earlier.

The overall mortality rate of 21 deaths did not differ in this group from the rate expected in three external reference populations or from that observed in two international comparison groups. Of the 21 deceased persons, 7 had cancer, compared with an expected 4.1 rate. Three of these deaths were due to stomach cancer, compared with an expected rate of 0.6. This expected rate was obtained from regional mortality data. One stomach cancer occurred among 148 individuals in the two comparison cohorts. If this particular study by Thiess et al. is carefully reviewed, a number of problems become obvious. One is the choice of the comparison groups. An additional problem is the fact that the number of deaths (19) is so small. We need additional studies with more participants and more carefully selected comparison groups to clarify the observations made by Thiess et al. Several such studies are in progress.

On the other hand, Hardell and Sandstrom (33) and Eriksson et al. (34) conducted two case-control studies in Sweden and reported an increased risk of soft-tissue sarcomas (cancers of soft-tissue) in men who were exposed to trichlorophenol or to phenoxy herbicides. In a third case-control study, investigators suggested that phenoxy herbicides and chlorophenols may also predispose to Hodgkin's lymphoma. Other sources however, provide little support for this theory (35).

Recently, Coggon and Acheson summarized the Swedish studies (36). They concluded: "Further research is urgently needed to confirm or refute these associations, to define the extent of the risk (if any) and to identify the carcinogen(s)." The Swedish studies could not be substantiated by Milham (37). Preliminary results from a case-control study in New Zealand have not indicated an excess risk of soft tissue sarcoma (38). We must kept in mind, however, that the mortality rate for soft-tissue sarcoma for U.S. males between the ages of 40 and 64 is extremely low and ranges from about 5 to 20 per million. This low incidence severely limits the power in some reported studies to detect such rare tumors.

The results of work by Honchar and Halperin (39) and Cook (40) of the United States supported the Swedish studies. Honchar and Halperin (39) noted that in four merged cohorts of exposed workers there were 105 deaths, 3 (2.9%) of which were due to soft-tissue sarcoma. On the basis of national death rates for men age 20 to 80, only 0.07% of the deaths due to soft tissue sarcoma would have been expected. After this was noted, however, another person in one of the four cohorts was found to have a soft-tissue sarcoma (40-42), which made a total of four soft-tissue sarcomas.

A review of tissue sections from these four tumors and from tumors of three additional cases gave some interesting results. Only two of the four cases with documented evidence of exposure and three additional cases, which did not have documented evidence of exposure, were confirmed to be soft tissue sarcomas (43). Thus, upon review by experts, not all of the tumors originally diagnosed as soft tissue sarcomas were found to be soft tissue sarcomas.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch005

Further studies will have to be conducted to determine whether exposure to these types of chemicals are associated with a higher incidence of soft tissue sarcomas.

All of these studies, in which associations between exposure to TCDD and an increased incidence of soft tissue sarcoma were reported, involved workers who had either manufactured these compounds or had applied the herbicide 2,4,5-T. In all of these instances, however, the workers were exposed not only to TCDD but also to either chlorinated phenols or to phenoxy herbicides.

General Population

Two poisoning outbreaks have occurred in the general population. One was in 1976 in Seveso, Italy, when a reaction vessel in a factory exploded (44,45). The chemicals in the vessel escaped into the air and descended as a cloud on the surrounding neighborhood. The chemicals were trichlorophenol, sodium trichlorophenate, ethylene glycol, sodium hyrdroxide, and TCDD. The population living adjacent to the plant was exposed to the cloud and later to contaminated vegetation. Two weeks after the incident, the people living in the contaminated area were evacuated. In this situation, the exposure levels in the cloud were probably quite high. A few vegetation samples collected within the first 2 weeks after the incident contained up to 15 ppm (mg/kg) of TCDD. Thus, significant exposures probably occurred during the first few weeks after the explosion. Within these first few weeks, 22 people (7 adults and 15 children aged 3 to 13) were hospitalized or admitted to outpatient clinics. A dermatitis that resembled first-and second-degree burns, with blisters and, later, crusts developed in these patients. In addition, lacrimation and the sensation of grit in the the eyes were reported, but conjunctivitis was not observed. These skin lesions healed within 2 to 3 months. Between 2 and 8 weeks after exposure, a number of patients developed blackheads and cysts of different sizes typical of chloracne. These lesions started to appear as the other skin lesions were fading.

In the first few days, gastrointestinal disturbances were also noted. A number of patients had slightly enlarged livers. In these cases, no evidence was detected of a peripherial neuropathy or of an effect on the central nervous system. The immune response in this population was within the normal range.

Since then, a number of health studies have been conducted in this population (46). A small percentage of the children in the area had chloracne. The number of cases ranged from 167-196, depending on whether very mild cases (which were not unequivocal chloracne) were included in the count and at what time these counts were made. None of these cases were as disfiguring as cases observed in some of the workers discussed earlier. Although liver problems, enlarged livers, and some abnormal results for liver function tests were reported, when the overall results of these studies were evaluated, no severe systemic health effects were noted (47,48).

Another episode in which a few people received substantial exposure to TCDD occurred in Missouri. In 1971, waste from the production of hexachlorophene at a manufacturing facility in

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch005

southwestern Missouri was mixed with salvage oil and sprayed on various sites throughout the State for dust control (11,49). This waste had high concentrations of TCDD. The source of the TCDD waste was a plant where 2,4,5-trichloro-phenol was made for the production of hexachlorophene. The poisoning episode was first discovered when a 6-year-old girl from Lincoln County, Missouri, was admitted to St. Louis Children's Hospital with an acute hemorrhagic cystitis (inflammation of the urinary bladder with bleeding). The child had played in the soil of a riding arena. Her first symptoms were headache, epistaxis (nose bleeds), diarrhea, and lethargy. She then had a hemorrhagic cystitis. Three other persons who had used the same riding arena complained of recurrent headaches, skin lesions, and polyarthralgia (pain in more than one joint). The concentration of TCDD in the soil of this arena was 30 ppm, or 30 mg/kg. The child's acute symptoms subsided after a time (50). Chloracne developed in two little boys, who had played in another contaminated riding arena, and adults associated with the arena complained of headaches.

A pilot study was conducted in a small group of people from another, more recently discovered, contaminated area in Missouri (51). This group's exposures were much lower. The soil contamination in most areas was generally less than 1 ppm of TCDD (mg/kg). The group consisted of 68 people who were possibly exposed and 35 people who were presumably not exposed. There were no statistically significant differences in the test results between the two groups.

Results of an additional study were reported recently (52). In this study 154 exposed and 155 unexposed persons were examined. The exposed persons had been living in a mobile home park in which 2,3,7,8-TCDD contaminated waste oil was sprayed for dust control. The TCDD contamination dated back to 1971 when TCDD-contaminated sludge wastes were taken from a hexachlorophene production facility (11,53). Levels of 2,3,7,8-TCDD up to 2.2 ppm (mg/kg) were still present more than ten years later. No increased clinical illness was noted in the group who had lived in the contaminated mobile home park. Some differences were noted, however, in the immunological responses between the two groups. All persons were given a skin test to measure delayed-type hypersensitivity. Half of the results in both groups could not be used because two of the four persons reading the skin tests apparently did not interpret the skin responses properly. Of the remaining participants, however, 11.8% in the exposed group and 1.1% in the control group were anergic (transient reduction or complete lack of reactivity to antigens or allergens). The relative anergy was noted in 35.3% of the exposed group versus 11.8% of the control group. The exposed group had a nonstatistical increased frequency of abnormal T-cell subset test results. Many parameters can affect the results of these tests, such as age, psychological stress, alcohol consumption, malnutrition, and infectious agents (54). It is not clear, in the absence of clinical disease, what these results mean. Further studies are needed to confirm these findings. Ratios of subsets of lymphocytes may be

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch005

affected by the total lymphocyte count. Apparently the exposed group had an elevated white blood cell count which was statistically significant.

The concentration of 2,3,7,8-TCDD has now been measured in the adipose tissue of 39 persons from Missouri with a history of residential (19), recreational (5), or occupational (15) exposure, and 57 persons in a control group. All participants had detectable levels of 2,3,7,8-TCDD, but the exposed group had significantly higher levels ($p < 0.001$). Levels in the controls ranged from 1.4-20.2 ppt (ng/kg), in the recreational group from 5.0-577 ppt (ng/kg), in the residential group from 2.8-59.1 ppt (ng/kg), and in the occupationally exposed group from 3.5-750 ppt (ng/kg). The geometric means for the different groups were: controls 6.4 ppt (ng/kg), recreational 38.9 ppt (ng/kg), residential 14.6 ppt (ng/kg), occupational 29.8 ppt (ng/kg). Residential exposure was defined as either living in close proximity to areas with 2,3,7,8 TCDD contaminated soil, or having evidence of contamination inside the home. Recreational exposure was defined of at least one time per week riding or caring for horses in 2,3,7,8-TCDD contaminated stable arenas. Occupational exposure was defined as working in a hexachlorophene production facility or at truck terminals where the grounds had been sprayed with 2,3,7,8 TCDD contaminated waste oil.(Patterson, D. G., Jr. Ph.D., Hoffman, R. E., Needham, L. L., Roberts, D.W., Bagby, J. R., Pirkle, J. L., Falk, H., Sampson, E. J., Houk, V. N. JAMA in press.) The preliminary findings suggest that people living in 2,3,7,8-TCDD contaminated areas may have slightly higher body burdens than the general populations. It is possible that this is unique to Missouri since 2,3,7,8-TCDD was mixed in an oil when applied to soil and riding arenas and has a greater bioavailability (55) than when mixed with soil from other areas (56). In the future these determinations will help in the assessment of exposure.

In addition, a morbidity study was recently completed (Ranch Hand study) on pilots who flew spraying missions in Vietnam and on other Air Force personnel. These members of the military were exposed to Agent Orange, a mixture of the herbicides 2,4-D and 2, 4,5-T. The 2,4,5-T was contaminated with 2,3,7,8-TCDD, (57). None of the findings were alarming. Findings differed only in the occurrence of skin cancers (mostly basal cell carcinomas), which were significantly higher in the Ranch Hand group when measured against the comparison group ($p = 0.03$). Thirty-one basal cell carcinomas occurred among the Ranch Hand group and 21 among the comparison group. These results, however, have not been corrected for geographic area, ethnic backgrounds, and excessive exposure to sunlight. Preliminary analysis of fertility and reproduction suggests a clustering of birth anomalies of the skin in Ranch Handers' children. In addition, the neonatal death rate ($p = 0.02$) was significantly increased for the Ranch Hand group. Before their exposure in Southeast Asia, the Ranch Hand group had 20 newborn offspring who died, and the comparison group had 17. After their service in Southeast Asia, however, the Ranch Hand group had 14 neonatal deaths and the comparison group had only 3. The Ranch

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch005

Handers also had significant deficits in two specific peripheral leg pulses and in all leg pulses, as a group. Ranch Handers, in addition, had elevated liver enzyme levels and lower cholesterol levels. More Ranch Handers were found to have hepatomegaly (enlarged liver) and verified histories of prior hepatic disease than their counterparts. Additional data analysis and follow-up of the Ranch Handers may clarify some of the preliminary findings made in this cross-sectional study. None of these findings, however, could be related to herbicide exposure because no specific "dose-response effect" could be shown.

The retrospective assessment of exposure in situations like Ranch Hand is extremely difficult, even if the environment is well defined and the exposure levels of a certain chemical are known. In an occupational situation, for instance, two people in the same environment can, for a variety of reasons, receive different doses. The reasons include variations in personal hygiene and in the ability to metabolize and excrete chemicals. For example, some investigators have found that workers who smoke have higher levels of chemicals in their body fluids than their nonsmoking counterparts.

In a retrospective study such as this, when the last exposure to Agent Orange was at least 12 years ago, it is difficult to assess what effect other, later insults may have had on the subjects' health. Furthermore, for purely statistical reasons, some differences will be found if many endpoints are examined. None of the findings in the Ranch Hand study have been confirmed in other studies.

The Centers for Disease Control recently completed a case-control study to determine if men who served in the U.S. military in Vietnam have been at an increased risk of fathering babies with serious congenital malformations. (58). Again, no striking findings were made, and it is not really clear whether this study should be more appropriately classified as a Vietnam experience study because of the great difficulties in appropriately determining exposure to 2,3,7,8-TCDD in Agent Orange.

The same can be said for other anecdotal reports that have linked exposure to the herbicide 2,4,5-T to a variety of undesirable health effects. Some of this information is summarized by Reggiani (44). Since most of the reports are anecdotal, and on reexamination could not be verified, they will not be reviewed here.

In conclusion, the most prevalent lesion in humans after acute exposure to 2,3,7,8-TCDD is a skin lesion referred to as chloracne. This skin disease may be accompanied by hirsutism and hyperpigmentation. After acute exposure to toxic levels, liver function may be impaired and a sensory neuropathy may be present. There may be complaints of weakness, weight loss, severe fatigue, and a general malaise. Many of these acute symptoms and signs revert to normal when exposure ceases. The chloracne is probably the most persistent lesion. No convincing chronic human health effects, other than chloracne, have been reported, nor is the dose of 2,3,7,8-TCDD known that would cause systemic illness or death in humans. Virtually no information is available on other PCDD congeners.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch005

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch005

Literature Cited

1. Kimbrough, R. D.; Falk, H.; Stehr, P. J. Toxicol. Environ. Health 1984, 14, 47-93.
2. Buser, H. R.; Bosshardt, H. P. J. Chromatogr. 1974, 90, 71-77.
3. Buser, H. R.; Bosshardt, H. P. J. Assoc. Off. Anal. Chem. 1976, 59, 562-69.
4. Buser, H. R.; Bosshardt, H. P. Mitt. Geb. Lecbensmittelunters 1978, 69, 191-99.
5. Kimbrough, R. D. Pathologist 1983, 37(9).
6. Czuczwa, J. M.; McVeety, B. D.; Hites, R. A. Chemosphere 1985a, 14, 623-26.
7. Czuczwa, J. M.; Niessen, F.; Hites, R. A. Chemosphere, 1985b, 14, 1175-79.
8. Schecter, A.; Ryan, J. J.; Lizotte, R.; Sun, W. F.; Miller, L.; Gitlitz, G.; Bogdasarian, M. Chemosphere 1985, 14, 933-37.
9. Ryan, J. J.; Lizotte, R.; Lau, B. P. Y. Chemosphere 1985a, 14, 697-706.
10. Ryan, J. J.; Schecter, A.; Lizotte, R.; Sun, W. F.; Miller, L. Chemosphere 1985b, 14, 929-32.
11. Carter, C. D.; Kimbrough, R. D.; Liddle, J. A.; Cline, R. F.; Zack, M. M.; Barthel, W. F.; Koehler, R. E.; Phillips, P. E. Science 1975, 188, 738-40.
12. Hoffmann, R.; Baader, E. W.; Bauer, H. J. Ind. Med. Surg. 1986 20, 286-90.
13. Baader, E. W.; Bauer, H. J. Ind. Med. Surg. 1951, 20, 286-90.
14. Behrbohm, P. Dtsch. Gesundheitswes. 14, 614-19.
15. Goldmann, P. J. Schwerste akute Chlorakne durch Trichlorophenol Zersetzungsprodukte. Arbeitsmed Sozialmed Arbeitshyg 1972, 7, 12-18.
16. May, G. Br J Ind Med 1973, 30, 276-283.
17. Zack, J.A.; Suskind, R.R. J. Occup. Med. 1980, 22, 11-14.
18. Thiess, A. M.; Frentzel-Beyme R.; Link, R. Am J Ind Med 1982, 3, 179-189.
19. Kimming, J.; Schulz, K. H. Dermatologica 1957, 115, 540-46.
20. Dugois, P.; Colomb, L. Lyon Med 1956, 88, 446-47.
21. Hofmann, H. Arch Exp Pathol Pharmakol 1957, 232, 228-30.
22. Oliver, R. M. Br J Ind Med 1975, 32, 49-53.
23. Jirasek, L.; Kalensky, J.; Kubec, K.; Pazderova, J.; Lukas, E.; Hautarzt 1976, 27, 328-333.
24. Pazderova-Vejlupkova, J.; Lukas, E.; Nemcova, M.; Pickova, J.; Jirasek, L. Arch Environ Health 1981, 36, 5-11.
25. Bleiberg, J.; Wallen, M.; Brodkin, R.; Applebaum, I.L. Arch Dermatol 1964, 89, 793-97.
26. Cam, S. Ann Derm Syph 1960, 87, 393-97.
27. Peters, H.A.; Gocmen, A.; Cripps, D.J.; Bryan, G.T.; Dogramaci, M. D. Arch Neurol 1982, 39, 744-49.
28. Poland, A.P.; Smith, D.; Metter, G.; Possick, P. Arch Environ Health 1971, 22, 316-27.
29. May, G. Br J Ind Med 1982, 39, 128-135.
30. Suskind, R. R.; Hertzberg, V. S. JAMA 1984, 251, 2372-80.

31. Frentzel-Beyme, R. Am J Epidemiol 1981, 114, 425.
32. Moses, M.; Lilis, R.; Crow, K. D.; Thornton, J.; Fischbein, A.; Anderson, H. A.; Selikoff, I. J. Am J Ind Med 1984, 5, 161-182.
33. Hardell, L., Sandstrom, A. Br J Cancer 1979, 39, 711-17.
34. Eriksson, M. H.; Hardell, O. L.; Axelson, O.; Berg, N. O.; Moeller, T. Br. J. Ind. Med. 1981, 38(1), 27-33.
35. Hardell, L.; Eriksson, M.; Lenner, P.; Lundgren, E. Br J Cancer 1981, 43, 169-176.
36. Coggon, D.; Acheson, E. D. Lancet. 1982, 1, 1057-59.
37. Milham S. Lancet 1982, 1, 1464-65.
38. Smith, A. H.; Fisher, D. O.; Giles, H. J.; Pearce, N. Chemosphere 1983, 12, 565-71.
39. Honchar, P. A.; Halperin, W. E. Lancet 1981, 1, 268-69.
40. Cook, R. R. Lancet 1981, 618, 3-14.
41. Cook, R. R.; Townsend, J. C.; Ott, M. G.; Silverstein, L. G.; JOM 1980, 22, 530-32.
42. Ott, M. G.; Holder, B. B.; Olson, R. D. J Occup Med 1980, 22, 47-50.
43. Fingerhut, M.A.; Halperin, W.E.; Honchar, P.A.; Smith, A.B.; Groth, D.H.; and Russell, W.O. Banbury Report. 1984, 18, 461-70.
44. Reggiani, G. J Toxicol Environ Health 1980, 6, 27-43.
45. Pocchiani, F.; Silano, V.; Zampieri, A. Ann NY Acad Sci 1979, 320, 311-20.
46. Tognoni, G.; Bonaccorsi, A. Drug Metab Rev 1982, 13, 447-469.
47. Reggiani, G. Arch Toxicol 1978, 40, 161-88.
48. Reggiani G. Pergamon Ser Environ Sci 1982, 5, 463-493.
49. Kimbrough, R. D.; Carter, C. D.; Liddle, J. A.; Cline, R. E.; Phillips, P. E. Arch Environ Health 1977, 32, 77-85.
50. Beale, M. G.; Shearer, W. T.; Karl, M. M.; Robson, A. M. Lancet 1977, 1, 748.
51. Centers for Disease Control. MMWR 1984, 33, 54-61.
52. Hoffmann, R. E.; Stehr-Green, P. A.; Webb, K. B.; Evans, R. G.; Kmetson, A. P.; Schramm, W. F.; Staake, J. L.; Gibson, B. B.; Steinberg, K. K. JAMA 1986, 255, 2031-38.
53. Stehr, P. A.; Forney, D.; Stein, G.; Donnell, H. D.; Falk, H.; Hotchkiss, R.; Spratlin, W. A.; Sampson, E.; Smith, S. J. Public Health Rep. 1985, 100, 289-293.
54. Vos, J. G. Immune suppression as related to toxicology. CRC, Crit. Rev. Toxicol., 1977, 67-101.
55. McConnell, E. E.; Lucier, G. W.; Rumbaugh, R. C.; Albro, P. W.; Harvan, D. J.; Hass, J. R.; Harris, M. W. Science, 1984, 223, 1077.
56. Umbreit, T. H.; Hesse, E. J.; Gallo, M. A. Science, 1986, 232; 497-499.
57. Lathrop, G. D.; Wolfe, W. H.; Albanese, R. A.; Moynahan, P. M. Brooks Air Force Base, Texas: USAF School of Aerospace Medicine, Aerospace Medical Division, 1984.
58. Erickson, J. D.; Mulinare, J.; McClain, P. W.; Fitch, T. G.; Levy, M. J.; McClearn, A. B.; Adams, M. J. JAMA 1984, 252, 903-12.

RECEIVED January 2, 1987

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch005

Chapter 6

2,3,7,8-Tetrachlorodibenzo-*p*-dioxin: Environmental Chemistry

Glenn C. Miller[1] and Richard G. Zepp[2]

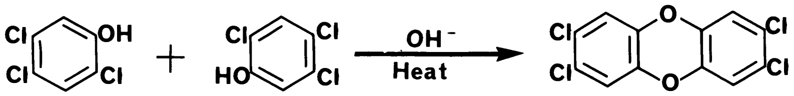
[1]Department of Biochemistry, University of Nevada—Reno, Reno, NV 89557
[2]Environmental Research Laboratory, U.S. Environmental Protection Agency, Athens, GA 30613

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch006

For molecules of its size and molecular weight, 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) is one of the most toxic, least water soluble, least volatile, and most resistant to thermal and biological transformations. The combination of these properties has presented a challenging problem in assessing human hazards ascribable to TCDD because, although exposure concentrations are often low, the time frame for transport and most degradative processes in the environment is generally long. Under normal environmental conditions the only transformation process that is fairly rapid for dioxins is photolysis.

This introductory chapter is designed to provide a general background on the environmental transformation and transport of TCDD, with emphasis on its environmental chemistry. It is not a complete review of the substantial amount of literature available on the topic, but is meant to set the stage for the following papers that discuss certain aspects of this subject in greater detail.

2,3,7,8-TCDD is a symmetrical molecule, generally synthesized by the condensation of two molecules of 2,4,5-trichlorophenol in the presence of base at high temperatures (1).



2,4,5-trichlorophenol 2,3,7,8-TCDD

An abbreviated list of its physicochemical properties is given in Table I. A more complete list of other properties is presented by

0097-6156/87/0338-0082$06.00/0
© 1987 American Chemical Society

Schroy and co-workers (2). Because of the extreme character of many of these properties, refinement of these values is an ongoing process. Reports on improved measurements of water solubility and vapor pressure were recently published (3,5).

Table I. Selected Chemical and Physical Properties of 2,3,7,8-TCDD

Property		Reference
Molecular weight	321.974	
Vapor pressure		
(25° C)	$7.4 \pm 0.4 \times 10^{-10}$ torr	(3)
(30° C)	3.46×10^{-9}	(2)
(54.6° C)	1.37×10^{-7}	(2)
(71° C)	1.19×10^{-6}	(2)
Melting point	305.0° C	(4)
Solubility in: (all at 25° C)		
water	19.3 nanograms/liter	(5)
benzene	570 milligrams/liter	(6)
methanol	10 milligrams/liter	(6)
acetone	110 milligrams/liter	(6)
Partition coefficient	1.4×10^{6}, 3.0×10^{8}	(7,8)
(octanol/water)	$(4.24 \pm 2.73) \times 10^{6}$	(5)
Molecular diffusivity:		
in air (calculated)	4.7×10^{-2} cm^2/s	(3)
in water (calculated)	5.1×10^{-6} cm^2/s	(3)
Photolysis quantum yields (313 nm)		
water-acetonitrile (90:10)	0.0022	(9)
hexane	0.049	(9)

Sources and Distribution

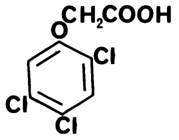
2,3,7,8-TCDD and related dioxins and dibenzofurans have come from two primary sources: (a) impurities in 2,4,5-trichlorophenol and related chemicals and (b) combustion processes. The products that have been manufactured from 2,4,5-trichlorophenol include herbicides such as 2-(2,4,5-trichlorophenoxy)propionic acid (silvex), 2,3,5-trichlorophenoxyacetic acid (2,4,5-T), and the disinfectant hexachlorophene (10). As discussed above, 2,3,7,8-TCDD is produced as a byproduct when 2,4,5-trichlorophenol is synthesized, and is found in significant concentrations in the synthesis residue (still bottoms). Chemical wastes containing 2,3,7,8-TCDD have been released directly into the environment. In some cases it has migrated from chemical waste dumps. For example, sediment samples taken near the Love

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch006

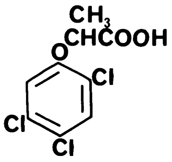
Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch006

Canal site in New York contained 0.9-312 ppb 2,3,7,8-TCDD, which presumably originated at a chemical waste dump (11). 2,3,7,8-TCDD was also spread over wide areas in Missouri when 2,4,5-trichlorophenol waste was incorporated into waste oil and used to control dust on roads, parking lots and horse arenas. The estimated volume of contaminated Missouri soil ranged from 150,000 to 400,000 cubic meters (12).

Agricultural and wartime use of trichlorophenol-based herbicides such as 2,4,5-T and silvex also has resulted in release of 2,3,7,8-TCDD at low concentrations in many countries.



2,4,5-T



silvex

These herbicides have been used extensively in silviculture for control of deciduous trees in conifer forests (13). As a military defoliant, 2,4,5-T was mixed with 2,4-dichlorophenoxy acetic acid (2,4-D), and an estimated 10 million gallons of this mixture was applied in South Vietnam under the name of Agent Orange. Subsequent analysis of human samples from South Vietnam has found 2,3,7,8-TCDD at elevated levels compared to samples from North Vietnam where Agent Orange was not used (14).

More recently, environmental contamination by chlorinated dioxins and furans released during combustion processes has been recognized. These substances are formed not only during incineration of chlorinated phenols, but also during combustion of materials in home fireplaces and municipal incinerators (10). Chlorinated dioxins and dibenzofurans also are formed during fires involving polychlorinated biphenyls (15).

Another potential source for the more toxic dioxin congeners is the photoreduction of the higher chlorinated dioxins, i.e. the octachloro and heptacholoro congeners, to the lower chlorinated isomers. There are conflicting results concerning which positions are photoreduced on the dioxin aromatic rings. If the 1,4,6,9-positions are most rapidly reduced, toxic tetra, penta and hexachloro isomers (2,3,7,8-substituted) may result. If, however, the lateral (2,3,7,8) positions are photoreduced, the resulting products are substantially less toxic. The available information on this question is mixed. Buser (16) observed that UV irradiation of solutions of octachlorodibenzo-p-dioxin (OCDD) resulted in preferential formation of 1,2,3,4,6,7,9-hepta-CDD, which indicates primary initial loss of the lateral (2,3,7,8) chlorines. This preferential loss of lateral chlorines was also observed by Dobbs and Grant (17). Alternatively, Lamparski and co-workers (18) report preferential loss of chlorine atoms from the 1,4,6,9-positions as the initial photoreduction of OCDD when irradiated on a wood surface. Cull and Dobbs (19) observed no preferential loss from either position. Both heptachloro

isomers were formed in similar yields. The reason for these differences remains unknown.

Because of their low water solubiliy and low volatility, 2,3,7,8-TCDD and related dioxins are predominantly sorbed on particles, particularly soils and sediments. Assessing exposure to these compounds requires the ability to predict their time-varying concentrations in those important environmental compartments. Both biological and non-biological transformation and transport processes must be considered, including hydrolysis, oxidation, reduction and phototransformation. For 2,3,7,8-TCDD, attempts have been made to model each process in several environmental systems, with varying degrees of success. Because of the toxicity of 2,3,7,8-TCDD, various model compounds have been utilized, particularly other chlorinated dioxins. Caution must be taken when extending these results to 2,3,7,8-TCDD, however, because this isomer is in many ways toxicologically and chemically unique. There is often only limited rationale for estimating the properties of 2,3,7,8-TCDD based on the behavior of other isomers, i.e. 1,4,6,9-TCDD.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch006

Biological Transformations

In contrast to the profound effect that 2,3,7,8-TCDD can have on biological systems, most biological systems have little impact on 2,3,7,8-TCDD. Early studies by Matsumura and Benezet (20) demonstrated that microbial metabolism was very slow if it occurred at all. Further work confirmed that although 2,3,7,8-TCDD is indeed relatively stable to microbial metabolism, two isolated microorganisms that gave water soluble metabolites were identified. In both aquatic sediment and terrestrial soil systems, metabolism of 2,3,7,8-TCDD was demonstrated. Addition of nutrients such as glucose, mannitol, and bactopeptone stimulated the transformation (21).

Substantial microbial degradation of 2,3,7,8-TCDD in oxic environments has not been demonstrated in field studies. Biodegradation was sufficiently slow that essentially all of the originally applied 2,3,7,8-TCDD in an experimental plot at Eglin Air Force Base in Florida in 1972 was still there in 1985 (22). Kimbrough and coworkers (23), estimated that the half-life for polychlorinated dibenzodioxins in soils was 10 years. Over a short (20 day) period, Pocchiari (24) did not observe increased rates of microbial degradation of 2,3,7,8-TCDD in organic-nutrient-amended soils from Seveso, Italy.

In a model ecosystem using aquatic sediment and lake water, metabolism played only a minor role in the loss of 2,3,7,8-TCDD (25). Volatilization was suggested to be the major mode of disappearance in samples incubated 39 days or more. The half-life under the conditions of the experiment in this model ecosystem was on the order of 600 days. Microbial metabolism appeared to be enhanced when microbial growth was stimulated; under such conditions metabolites were observed. One organism which can degrade 2,3,7,8-TCDD at an appreciable rate is the white rot fungus, *Phanerochaete chrysosporium* (26). The ability of this fungus to metabolize 2,3,7,8-TCDD was suggested to be dependent on an extracellular ligin-degrading enzyme system.

The lack of appreciable metabolism of 2,3,7,8-TCDD in aerobic environments can in part be explained by the high oxidation state of

the molecule. The six electron withdrawing atoms on 2,3,7,8-TCDD reduce the ease of oxidative metabolism. Although the available information is not substantial, reduction may be as important as oxidation. Evidence exists that 2,3,7,8-TCDD may be slowly degraded by anaerobic microorganisms in an organic matrix used for secondary treatment of chlorophenolic wastewaters (27).

Further support that reduction may be important for 2,3,7,8-TCDD is provided by the observation that it undergoes reduction to the lower chlorinated isomers in electron impact mass spectrometry when hydrogen is used as the gas chromatographic carrier gas (28). In addition, degradation of 2,3,7,8-TCDD in basic solutions of polyethyleneglycol and oxidants actually appears to occur by reductive processes, since the products are the lower chlorinated dioxins (29).

Abiotic Transformation

Oxidation, Hydrolysis and Reduction. 2,3,7,8-TCDD is not susceptible to hydrolysis at environmental temperatures (3) and is highly stable to chemical oxidation. TCDD exhibits thermal stability at environmental temperatures. Indeed, the thermal stability of TCDD is such that temperatures of 1400°F to 2400°F are used for efficient (>99.9999%) degradation of 2,3,7,8-TCDD from contaminated soils (30). Reaction rates with hydroxyl radicals have not been measured, although Podoll and co-workers (3) estimated the vapor phase rate constant to be on the order of 3×10^{8} $M^{-1}s^{-1}$. Using average OH radical concentrations to be 3×10^{-15} M, a half life of 200 hours was estimated for oxidation of TCDD in the atmosphere.

Photodegradation. Of all the possible natural degradation mechanisms, photodegradation has been suggested to be the most significant (3,9,10,27). 2,3,7,8-TCDD undergoes rapid degradation when exposed to ultra-violet light in the presence of a hydrogen (reducing) source. The photochemistry of dioxins has been reviewed by Choudhry and Hutzinger (31) and Esposito and coworkers (27).

Dulin and coworkers (32) have measured the quantum yield for photolysis of 2,3,7,8-TCDD in an acetonitrile/water mixture and in hexane (Table I). The more than 20-fold difference in quantum yield confirms either the need for a hydrogen source for efficient photolysis, or that the reaction pathway is substantially altered on going to a largely water solvent. The absorption spectra of 2,3,7,8-TCDD in acetonitrile shows a strong band centered at 309 nm with an extinction coefficient of 7020 $M^{-1}cm^{-1}$, whereas in hexane the band is centered at 304 nm with extinction coefficient of 5640 $M^{-1}cm^{-1}$. For sunlight photolysis, the higher quantum yield in hexane may be partially offset by reduced spectral overlap of 2,3,7,8-TCDD with the solar spectrum. In hexane, under identical conditions, the quantum yield for photolysis of 2,3,7-trichlorodibenzo-p-dioxin was 0.20, which was over four times higher than for 2,3,7,8-TCDD. Using the photolysis quantum yield, Podoll and coworkers (3) determined that the photolysis half-lives in surface waters varied from 21 hours in summer to 118 hours in winter.

Choudhry and Webster (33) also found low quantum yields for photolysis of 1,2,3,4,7-pentachorodibenzo-p-dioxin and 1,2,3,4,7,8-hexachlorodibenzo-p-dioxin of 9.8×10^{-5} and 1.1×10^{-4}, respec-

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch006

tively in water:acetonitrile (4:6) solutions. Both of these solvents are poor hydrogen donors, evidenced by the low quantum yields. Assuming that the quantum yields are the same in pure water, half lives of both compounds are 15 and 6 days, respectively, for the compounds in summer sun at 40° N. Botre' and coworkers (39) found that 2,3,7,8-TCDD was most rapidly lost in micelles of 1-hexadecylpyridinium chloride ($t_{1/2}$ 4 hrs) compared to micelles of sodium dodecyl sulfate ($t_{1/2}$ 8 hrs) and methanol ($t_{1/2}$ 18 hrs). Unfiltered mercury lamps were used in these studies.

Nestrick and coworkers (34) observed a striking difference in the photolysis rates of a series of tetrachlorinated dibenzo-*p*-dioxins. 2,3,7,8-TCDD underwent the most rapid photolysis in hexadecane. The rate of photolysis was 148 times more rapid than that of the 1,4,6,9-TCDD isomer when exposed to light from an RS sunlamp. Other TCDD isomers had relative photolysis rates varying between 2.7 times slower (1,3,7,8-TCDD) to 24 times slower (1,3,6,9-TCDD). The problem with relating these results to outdoor conditions is that the overlap of the absorption spectra of each of the isomers with the emission spectra of the RS sunlamp was not considered (35). 2,3,7,8-TCDD has an absorption band at 310 nm (extinction coefficient of 5590) in chloroform (36). This wavelength is also near the most intense emission band (314-316 nm) of the RS sunlamp between 290 nm and 320 nm (34). Other tetrachlorinated isomers have absorption maxima at wavelengths different than 2,3,7,8-TCDD. Even with this consideration, the data support evidence by others that photolysis of TCDD isomers is most rapid for those substituted on the lateral (2,3,7,8) positions.

Alternatively, Nestrick and co-workers (34) showed that 2,3,7,8-TCDD was the most stable of the 22 TCDD isomers examined on glass. 1,4,6,9-TCDD was the second most stable TCDD isomer, with a relative half-life that was 0.099 times that of 2,3,7,8-TCDD. The isomer most photoreactive on the glass surface, 1,2,3,9-TCDD, exhibited a half-life that was 0.014 times that of 2,3,7,8-TCDD. RS sunlamps also were used in this study, and no correction was provided for different absorption spectra of the various isomers. Although quantum yields would be more interpretable, it is nonetheless surprising that 2,3,7,8-TCDD photodecomposed the most rapidly in solution, but most slowly when exposed to light on a glass surface.

Molecular orbital calculations of 2,3,7,8-TCDD and dibenzo-*p*-dioxin indicate that excitation of either molecule results in promotion of an electron into an anti-bonding orbital with electron density primarily located on the 2,3,7,8 positions, with only a small percentage on the 1,4,6,9 positions (37). The primary photoproducts of 2,3,7,8-TCDD in organic solvent has been suggested to be the 2,3,7-trichloro isomer and the dichloro isomers (38). These photoproducts also appear to photodegrade faster than 2,3,7,8-TCDD. No other photoproducts have been identified from the photolysis of 2,3,7,8-TCDD, except for a "yellow, non-volatile gum" that was formed after extended exposure of methanolic solutions to fluorescent lamps (38).

Alternatively, a recent study by Dulin and coworkers (9) found no photoproducts for 2,3,7,8-TCDD in either water or hexane, despite a careful analysis of the reaction solution for 2,3,7-trichlorodibenzo-*p*-dioxin. They suggested that cleavage of one Ar-O bond is an alternative photochemical pathway which may predominate during 2,3,

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch006

7,8-TCDD photolysis. This mechanism would not result in chlorine replacement by hydrogen and would account for their finding that lower chlorinated dioxins were not produced in detectable quantities.

All of the studies to date have consistently demonstrated that the photolysis rate of 2,3,7,8-TCDD is dependent on the chemical environment in which it resides. Organic solvents dramatically accelerate the photolysis rate. Crosby and coworkers (38) found that, although the half-life of 2,3,7,8-TCDD in methanol under fluorescent lamp irradiation was approximately 3 hours, irradiation on either a Norfolk sandy soil for 96 hours, or as a thin dry film on glass for 14 days showed no loss of 2,3,7,8-TCDD. Wipf and coworkers (39) state that the photolysis rate of 2,3,7,8-TCDD on vegetation is increased by a factor of 25 when olive oil or arachis oil is used as a hydrogen source.

Photolysis of 2,3,7,8-TCDD on soil surfaces has been most studied to date by Liberti and coworkers (40). Their goal was to find methods to accelerate the loss of 2,3,7,8-TCDD from contaminated soil in Seveso, Italy. Unfiltered, high pressure mercury lamps were used in these studies. An important finding in this work is that a temperature dependence on the photolysis rate of 2,3,7,8-TCDD on glass plates was observed. At 23° C the half-life was approximately 50 hours; at 30° C the half-life was approximately 9 hours. Increased volatilization may have accounted for part of the greater loss at the higher temperature. Most significantly, under controlled conditions at 1 cm depth in soils, nearly 100% of the 2,3,7,8-TCDD was lost after nine days of exposure to summer sunlight, with an added hydrogen donor of 1:1 v/v solution of xylene and ethyl oleate. In addition, 40-60% was lost in the "lower" layers, presumably 2-3 cm depth. The surprisingly high loss in the lower layers was suggested to be due to either diffusion of 2,3,7,8-TCDD in the oleate layer or to a "photolytic reaction occuring through radicals."

Crosby and Wong (41) examined the sunlight photolysis of 2,3,7,8-TCDD on glass surfaces using various 2,4,5-T formulations, including Agent Orange, the herbicide Esteron and Esteron formulation without 2,4,5-T. In sunlight, photolysis of 2,3,7,8-TCDD in Agent Orange resulted in nearly 60% loss of 2,3,7,8-TCDD in 6 hours. In the presence of the herbicide, Esteron, 70% was lost in 6 hours, and nearly 90% was lost in 2 hours of sunlight exposure in Esteron formulation without 2,4,5-T. 2,3,7,8-TCDD photolysis loss in Agent Orange also was examined on soil and rubber plant leaf surfaces. Loss was again rapid on the plant surface with 70 to 90% loss in 6 hours. On soils, photolysis was substantially slower and more than 80% of 2,3,7,8-TCDD remained after 6 hours of exposure. Rapid initial loss from the soil was followed by a pronounced decrease in the photolysis rate constant. On 2,3,7,8-TCDD contaminated soils treated with olive oil, photodecomposition also was observed in experiments at Seveso, Italy, although substantial data scatter was apparent (42).

Gebefugi and coworkers (43) irradiated 2,3,7,8-TCDD sorbed on silica gel with pyrex filtered high pressure mercury arc radiation and found that only 8% remained following 18 days of exposure. They concluded that photochemical decomposition of 2,3,7,8-TCDD in sunlight is likely in soils, even without an added organic solvent.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch006

Plimmer (44) also found that 63% of 2,3,7,8-TCDD degraded on silica gel exposed to 20 hours of sunlight and produced a polar product. Only 68% of the original radioactivity was recovered after the 20-hour exposure, suggesting that volatility may be a factor in the loss. On soils, no difference was observed between exposed and non-exposed areas, suggesting that soil exerts a protective effect against the photolysis of 2,3,7,8-TCDD.

Exner and coworkers (45) have recently described a process of in-place detoxication of dioxin-contaminated soil using an unfiltered 450 watt mercury arc lamp. The goal of this work was to determine whether dioxin contaminated soils could be treated by exposure to intense ultraviolet light. As in other studies, an organic solvent greatly accelerated the process. Using Times Beach, Missouri soils, which were heavily contaminated, they demonstrated substantial reduction in the amount of 2,3,7,8-TCDD on the soils when various soil amendments were used. The soil depth in these experiments ranged from 4 to 8 mm and contained: (a) ethyl oleate/o-xylene sprayed at 0.35 L/m^2 (27 hour exposure gave 81% removal); (b) ethyl oleate/water surfactant (75-hour exposure gave 68% removal); and (c) aqueous surfactants at 2% w/w of the soils (7 hour exposure gave 63% removal). In the last experiment (3), irradiation of dry soils for 7 hours gave no detectable loss of 2,3,7,8-TCDD, and soils sprayed only with water and irradiated for 7 hours showed only 6% loss of 2,3,7,8-TCDD. Mixing and rewetting the soils with the organic solvent at 30-minute intervals during irradiation resulted in greater loss of 2,3,7,8-TCDD. In each of the experiments using aqueous surfactants, the soils were rewetted during the irradiation. The primary conclusion of this research was that in-place destruction of 2,3,7,8-TCDD occurs readily in the presence of 0.3-3% w/w levels of common agricultural surfactants. It also implied that solubilization and transport to the air/soil surface may be a necessary step for degradation of 2,3,7,8-TCDD by light.

Measurement of the vapor phase photolysis of 2,3,7,8-TCDD is experimentally a major challenge due to the low volatility. Podoll and coworkers (3), however, have estimated the half life of 2,3,7,8-TCDD in the vapor phase to be 55 minutes. This estimate assumed that the quantum yield is the same as in hexane and is invariant with wavelength, that the spectral properties are the same as in solution, and that the mechanisms for transformation are the same. This photolysis half-life is approximately 200 times smaller than that expected for reaction with hydroxyl radical. However, both vapor phase photolysis and reactions with OH radicals would be unimportant if TCDD is predominantly sorbed on particles in the atmosphere. Experimental verification of these estimates is needed, because atmospheric transformation may be an important fate of 2,3,7,8-TCDD and other dioxins which enter the atmosphere from combustion or from wind erosion of contaminated surface soils.

In summary, published investigations to date indicate that:

1. 2,3,7,8-TCDD undergoes relatively rapid photolysis in sunlight when dissolved in solution.
2. The rate of photolysis is dependent on the chemical microenvironment; organic solvents greatly accelerate the photolysis.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch006

3. Photolysis of 2,3,7,8-TCDD in soils is slow, and was not observed in several studies: addition of an organic solvent to the soils promotes the photolysis.
4. Information on how soil properties affect the photolysis is lacking. Also, the depth dependence of photolysis is not well understood.
5. Successful modeling of 2,3,7,8-TCDD photolysis in soils requires additional basic information on the significance of photolysis in relation to other fate and transport processes.

Transport Processes

2,3,7,8-TCDD and related compounds are transported while sorbed on particles, while dissolved in water or other solvents, or while dispersed in the vapor state. If photolysis is the primary pathway for detoxication of TCDD, transport to sunlight exposed areas may be the limiting factor for degradation, especially in soils where vertical transport of 2,3,7,8-TCDD is exceedingly slow. Its extremely low water solubility and vapor pressure combine to give a rate of movement expressed in cm/year in soils (22), although added organic solvents can greatly increase the mobility of TCDD. Consequently, estimating the rate of movement of 2,3,7,8-TCDD in soils requires detailed data on the soil characteristics and the presence of solvents or oils.

The reported log octanol-water partition coefficient (K_{ow}) for 2,3,7,8-TCDD varies from 6.15 to 8.48 (3,7,8). The magnitude of this value, in addition to the low water solubility, suggests that it will be primarily associated with particles in sediment and soil, with a vanishingly small fraction in the non-sorbed phase. This was demonstrated by Jackson and coworkers (46) who examined the solubility of 2,3,7,8-TCDD in ten soils. They observed that the soil partition coefficient for ten soils ranged from 0.4×10^5 to 45.8×10^5. Although 2,3,7,8-TCDD would move only very slowly in these soils, if oils or other solvents were present, sorption coefficients may be much lower and thus movement significant.

Volatilization of 2,3,7,8-TCDD from soils is expected to be very slow, due to the low vapor pressure and high octanol-water partition coefficient. Mill (47) estimates that the half-life for vaporization of 2,3,7,8-TCDD from soils will range from many months to years, in the absence of intervening transformation processes. Due to this rather substantial period, and the observed relatively rapid photolysis on soils, vaporization from soils may not be a significant source of 2,3,7,8-TCDD in the atmosphere. The main source of cholorinated dibenzodioxins in the atmosphere is probably municipal and industrial incineration (10). Because of large partition coefficients, dioxins in the atmosphere are likely associated predominantly with particles. Further information is needed, however, to firmly resolve the physical state of dioxins in the atmosphere.

In the top several centimeters of soil, photolysis, volatilization, mass transport in water either dissolved, sorbed on particles, or complexed with other molecules, and bioturbation are potential processes that affect chemical behavior. Freeman and Schroy (22) have developed a model for movement of 2,3,7,8-TCDD in soils based

In Solving Hazardous Waste Problems; Exner, J.;
ACS Symposium Series; American Chemical Society: Washington, DC, 1987.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch006

on volatilization as the predominant transport process. To simulate the effect of temperature on the transport of 2,3,7,8-TCDD in soil a time dependent energy balance model was constructed. Using this model they calculated the expected movement of 2,3,7,8 TCDD at a site at Eglin Air Force Base that had been intentionally contaminated 12 years previously at a depth of 11 cm. Careful analysis of the various depths of the soil indicated that 2,3,7,8-TCDD had dispersed approximately 10 cm during the 12-year period. Agreement was found between the model results and the observed depth profile.

Podoll and coworkers (3) have redetermined the vapor pressure of 2,3,7,8-TCDD. Using this information together with the aqueous solubility, octanol/ water partition coefficient and photolysis quantum yields, investigators have estimated the half-lives for movement and transformation of 2,3,7,8-TCDD in water and air. Even though the vapor pressure (P) of 2,3,7,8-TCDD is low, the water solubility (S) is also very low and the Henry's law constant is therefore significant, and allows vaporization from water.

$$H = P/S = 7.4 \times 10^{-10} \text{ torr}/6 \times 10^{-11} \text{ M}$$

$$= 12 \text{ torr M}^{-1}$$

Using a two film model, the volatilization half-lives for 2,3, 7,8-TCDD were estimated to be 32 and 16 days, respectively, in 200 cm deep ponds and rivers.

Concluding Comments

2,3,7,8-TCDD is the most toxic of the dioxins and remains a primary concern for exposure to humans. Although combustion sources of this compound are now readily accepted, waste contaminated soils and waste dumps remain a costly problem.

The extensive amount of research on this compound has indicated that 2,3,7,8-TCDD is both remarkably stable and is almost exclusively associated with particulate material. Because of the low water solubility and high sorption on soils, it moves only very slowly in natural soils. Human exposure to 2,3,7,8-TCDD in soils is thus a function of movement and degradation (primarily photochemical) on soil particles. Enhanced movement of 2,3,7,8-TCDD in soils also contaminated with oils and/or solvents potentially can increase concentrations near the surface, and this process, along with photolysis on particles, requires further investigation.

Literature Cited

1. Nestrick, T. J.; Lamparski, L. L.; Stehl, R. H. Anal. Chem. 1979, 51, 2273-2281.
2. Schroy, F. M.; Hileman, F. D.; Cheng, S. C. Chemosphere 1985, 14, 877-880.
3. Podoll, R. T.; Jaber, H. M.; Mill, T. Environ. Sci. Technol. 1986, 20, 490-492.
4. Boer, F. P.; van Remoortere, F. P.; Muelder, W. W. J. Am. Chem. Soc. 1972, 94, 1006-1007.
5. Marple, L.; Runck, R.; Throop, L. Environ. Sci. Technol. 1986, 20, 180-182.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch006

6. Esposito, M. P.; Teirnan, T. O.; Dryden, F. E. "Dioxins", U.S. EPA, Office of Research and Development, Cincinnati, Ohio, EPA-600/2-80-197, 1980.
7. Kenaga, E. E. Environ. Sci. Technol. 1980, 14, 553-556.
8. Surna, L. P.; Hodge, P. E.; Webster, G. R. B. Chemosphere 1984, 13, 975.
9. Dulin, D.; Drossman, H.; Mill, T. Environ. Sci. Technol. 1986, 20, 72-77.
10. Hutzinger, O.; Blumich, M. J.; Berg, M. V. D.; Olie, K. Chemosphere 1985, 14, 581-600.
11. Smith, R. M.; O'Keefe, P. W.; Aldous, K. M.; Hilker, D. R.; O'Brien, J. E. Environ. Sci. Technol. 1983, 17, 6-10.
12. Yanders, A. F.; Kapila, S.; Schreiber, R. J. In "Chlorinated Dioxins and Dibenzofurans in Perspective"; Rappe, C.; Choudhary, G.; Keith, L. H., Eds., Lewis Publishers, Chelsea, Mihigan, 1986; pp. 237-239.
13. Ingelog, I. In "Chlorinated Phenoxy Acids and Their Dioxins"; Ramel, C., Ed.; Swedish Natural Science Research Council: Lund, Sweden, 1978; pp. 240-254.
14. Schecter, A. J.; Ryan, J. J.; Gross, M.; Weerasinhe, N. C. A.; Constable, J. D. In "Chlorinated Dioxins and Dibenzofurans in Perspective"; Rappe, C.; Choudhary, G.; Keith, L. H., Eds.; Lewis Publishers: Chelsea, Michigan, 1986; pp. 35-50.
15. The Trace Chemistries of Fire--A Source of and Routes for the Entry of Chlorinated Dioxins into the Environment, The Chlorinated Dioxin Task Force, The Michigan Division of Dow Chemicals, U.S.A.; 1978.
16. Buser, H. R. J. Chromatography 1976, 129, 303-307.
17. Dobbs, A. J.; Grant, C. Nature 1979, 278, 163-165.
18. Lamparski, L. L.; Stehl, R. H.; Johnson, R. L. Environ. Sci. Technol. 1980, 14, 196-200.
19. Cull, M-R.; Dobbs, A. J. Chemosphere 1984, 13, 1984.
20. Matsumura, F.; Benezet, H. J. Environ. Health Perspect. 1973, 5, 253-258.
21. Matsumura, F.; Quenson, J.; Tsushimoto, G. In "Human and Environmental Risks of Chlorinated Dioxin and Related Compounds"; Tucker, R. E.; Young, A. L.; Gray, A. P., Eds.; Plenum Press: New York, 1983, pp. 191-221.
22. Freeman, R. A.; Schroy, J. M. Chemosphere 1985, 14, 873-876.
23. Kimbrough, R. D.; Falk, H.; Stehr, P.; Porter, C.; Fries, G. Health Implications of 2,3,7,8-TCDD Contamination of Residential Soil Center for Disease Control, Atlanta, 1983.
24. Pocchiari, F. In "Chlorinated Phenoxy Acids and Their Dioxins"; Ramel, C., Ed.; Ecol. Bull. Stockholm 1978, 27, 67-70.
25. Ward, C. T.; Matsumara, F. Arch. Environ. Cont. Toxicol. 1978, 7, 349-357.
26. Bumpus, J. A.; Tien, M.; Wright, D.; Aust, S. D. Science 1985, 228, 1434.
27. Esposito, M. P.; Tiernan, T. O.; Dryden, F. E. "Dioxins", EPA-600/2-80-197, U.S. Environmental Protection Agency, Cincinnati, Ohio; 1980.
28. Lau, B. P.-Y.; Sun, W.-P.; Ryan, J. J. Chemosphere 1985, 14, 799-802.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch006

29. Tundo, P.; Facchetti, S.; Tumiatti, W.; Fortunati, U. G. Chemosphere 1985, 14, 403-410.
30. Kleopfer, R. D.; Haxel, R. H.; Freestone, F. J.; des Rosiers, P. E. In "Chlorinated Dioxins and Dibenzofurans in Perspective"; Lewis Publishers; Chelsea, MI 1986; pp. 511-530.
31. Choudhry, G. G.; Hutzinger, O. Residue Rev. 1982, 84, 113.
32. Dulin, D.; Drossman, H.; Mill, T. Environ. Sci. Technol. 1986, 20, 72.
33. Choudhry, G. G.; Webster, G. R. B. Chemosphere 1985, 14, 9.
34. Nestrick, T. J.; Lamparski, L. L.; Townsend, D. I.; Anal. Chem. 1980, 52, 1865.
35. Zepp, R. G. In: "Handbook of Environmental Chemistry", Vol. 2, Part B; Hutzinger, O., Ed.' Springer-Verlag: Berlin; 1980, pp. 19-41.
36. Pohland, A. E.; Yang, G. C. J. Agr. Food Chem. 1972, 20, 1093.
37. Miller, G. C.; Sontum, S.; Crosby, D. G. Bull. Environ. Contam. Toxicol. 1977, 18, 611.
38. Crosby, D. G.; Wong, A. S.; Plimmer, J. R.; Woolson, E. A. Science 1971, 173, 748.
39. Wipf, H. K.; Homberger, E.; Neuner, N. Schlemlker In "Dioxin: Toxicological and Chemical Aspects"; Cattabeni, F.; Cavallaro, A.; Galli, G. Eds.; S. P. Medical and Sci. Books, 1978, p. 201.
40. Botre, C.; Memoli, A.; Alhaique, F. Environ. Sci. Technol. 1978, 12, 335.
41. Liberti, A.; Brocco, D.; Allegrini, I.; Cecinato, A.; Possanzini, M. Sci. Total Environ. 1978, 10, 97.
42. Crosby, D. G.; Wong, A. S. Science 1977, 198, 1337.
43. Crosby, D. G. In "Disposal and Decomtamination of Pesticides"; Kennedy, M. V., Ed.; American Chemical Society Symposim Series, No. 73. 1978, p. 1.
44. Gebefugi, I.; Baumann, R.; Korte, F. Naturwissenschaften 1977, 64, 486.
45. Plimmer, J. Bull. Environ. Contam. Toxicol. 1977, 20, 87.
46. Exner, J. H.; Alperin, E. S.; Groen, A., Jr.; Morren, C. E. Hazardous Waste 1984, 1, 217.
47. Jackson, D. R.; Roulier, M. H.; Grotta, H. M.; Rust, S. W.; Warner, J. S. In "Chlorinated Dioxin and Dibenzofurans in Perspective", Rappe, C.; Choudhary, G.; Keith, L. H., Eds.; Lewis, Publishers: Chelsea, MI, 1986, pp. 185-200.
48. Mill, T. In "Dioxins in the Environment", Damrin, M.; Rodgers, P. W., Eds.; Hemisphere Publishing Co., 1985, pp. 173-193.

RECEIVED January 16, 1987

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch006

Chapter 7

Persistent Toxic Organic Waste: Is Destruction Necessary?

Douglas J. Hallett

ELI—Eco Logic Inc., Rural Route 2, Acton, Ontario L7J 2L8, Canada

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch007

Persistent toxic chemicals are now ubiquitous throughout the global biosphere. Environmental processes such as long-range transport through the atmosphere play a major role in creating this global dispersion. They are also the major exposure route to the human species through the terrestrial food web. Hazard and risk assessment associated with persistent contaminants must focus on dynamics throughout the whole ecosystem and total human exposure. Best available or best practicable treatment processes for wastes that contain persistent contaminants must be evaluated in terms of ecosystem exposure rather than providing diversions. Examples include the diversion of contaminants from an aquatic effluent into sewage sludge that is then either incinerated at low temperature or land farmed; both of these methods allow volatilization into atmosphere. The adoption of loading limits versus acceptable concentrations derived by dilution of persistent toxic substances is necessary. This paper considers the chemical dynamics of persistent organochlorine contaminants in the Great Lakes ecosystem, the major sources, present waste treatment, and the total ecosystem exposure to the human species.

0097-6156/87/0338-0094$06.00/0
© 1987 American Chemical Society

Risk is a function of two variables: toxicity and exposure. This paper concerns not only environmental exposure but ecosystem cycling and ecosystem or true multi-media exposure for persistent organic toxic substances such as chlorinated dibenzo-p-dioxins (PCDD), chlorinated dibenzofurans (PCDF), PCBs, and other chlorinated structures. Although bioaccumulation due to the lipophilicity of organochlorines is important, it is not the sole factor to consider in assessing exposure. If one considers the chemical properties of these molecules to persist, and that these chemicals cycle between water and sediment, water and the atmosphere, and between soil, air and water, one can then begin to understand the multi-media exposure and risk that occurs today. Only then can the total risk for one compound (from any source such as a disposal process be mathematically estimated based on non-occupational ecosystem exposure. This risk estimation is key to understanding and prioritizing dioxin types and sources in the environment and deciding on remedial strategies.

The Great Lakes Sources And Pathways

The Great Lakes ecosystem is extremely sensitive as well as being relatively polluted with persistent toxic substances. Why? In 1977 our limited understanding led us to believe that the high levels of PCBs in the Great Lakes system were due to the vast number of sources of pollution coming from the eight industrial Great Lakes States and the Province of Ontario in Canada. However, experience with phosphate management in the Great Lakes and a growing knowledge of the physical limnology of these large, deep lakes, which exhibit high surface to volume ratios and extremely long retention times, has revealed that these properties allow the lakes to reach such high levels of persistent contaminants. The high surface to volume ratio allows the lakes to capture large quantities of persistent substances from long range atmospheric transport which brings contaminated rain. Examples include polychlorinated dibenzofurans and dioxins, which along with PCB and toxaphene are found in the sediments and fish of Siskiwit Lake, a remote lake found on Isle Royale, in north central L. Superior. This island has neither industry, agriculture, nor logging (1,2). The deposition rate of chlorinated dioxins and dibenzofurans on this island matches the deposition rate in L. Superior. The common source of these contaminants for both lakes is the atmosphere, which accounts for up to 85 % of the PCB and 100 % of the toxaphene entering L. Superior (3).

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch007

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch007

It is now obvious that atmospheric transport of persistent toxic organic substances is the major pathway between ecosystems. For dioxin, volatilization of residues from contaminated soils was first noted as a concern at Seveso, Italy (4). The National Research Council of Canada reported that atmospheric emissions were the major source of chlorinated dioxins in the Canadian environment (5). A recent Ontario report estimates that from 8 - 10 kg of 2,3,7,8 -TCDD equivalents enter the Ontario environment annually from combustion of municipal refuse and sewage sludge and that all other combustion sources contribute from 20 - 50 kg annually (6). The only other major source considered was from the use and disposal of chlorinated phenols.

In the Great Lakes we have observed some dramatic trends in persistent residues, particularly in L. Ontario. Spatial trends in sediment have indicated that industrial centres in the lower Great Lakes such as L. Erie and L. Ontario have contributed vast quantities of PCBs and lead into the water.

Temporal trends through time show us a confusing situation. On first glance one can see a dramatic improvement in L. Ontario residues of PCBs, mirex, and dioxin in wildlife and fish between 1970 and 1980 (7). However, from 1980 to 1984 the downward trend stopped for all of these residues. On examining the sediment record at the source of L. Ontario, the Niagara River, we find a deposition rate for PCBs and mirex from 1950 to 1960 at approximately the present day rate. However, at about 1960 a tremendous pulse of PCBs, mirex, chlorobenzenes, and hexachlorobutadiene is shown in the sediment record. The deposition rate tapers off to 1970 when it plateaus at about the 1950's rate (8). The water in the Niagara R. still contains PCB, mirex, chlorinated dioxins, and dibenzofurans. The sources are most likely leachate from the series of landfills in the area containing over 250 million tons of chlorinated waste (9). The phenomenon of rapid declines in industrial areas is quite common in the Great Lakes basin and likely reflects either controlled effluents or lower production of the key persistent contaminants.

If we look at L. Superior, which is far less industrialized than L. Ontario, we find that there has been a slow decline in PCB residues found in fish and wildlife. More importantly, residue levels are now similar to those found in the industrialized Great Lakes. This phenomenon is more characteristic of the global trends (10),(11).

In the Great Lakes region we have begun to follow PCB with mass balance or input-output models. Eisenreich et al, (3) has shown such a model for L.Superior. (Table I).

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch007

There is a flux occurring both downwards from the atmosphere and upwards again from the surface-water interaction. Mackay and Paterson (12,13) have considered this phenomenon with fugacity models. Molecules with lower vapour pressure tend to be more strongly sorbed to aerosols and so are more susceptible to washout in precipitation. PCBs such as represented in the mixture Aroclor 1254 have a vapour pressure of 7 x 10^{-5} mm Hg (14,15). These will be more readily washed out of the atmosphere than lesser chlorinated biphenyls such as those represented in the mixture Aroclor 1241 with a vapour pressure of 4 x 10^{-4} mm Hg. This same property allows for the volatilization of chlorinated biphenyls particularly with 3 and 4 chlorines per molecule during precipitation free periods. (13). Since the vapour pressure varies with temperature, winter seasons with lower temperatures and greater precipitation may encourage an increased downward flux wheras summer seasons would encourage a more upward flux.

Table I. Model of PCBs in Lake Superior

INPUT	$Kg \cdot yr^{-1}$	OUTPUT	
ATMOSPHERE	6,600-8,300	St. Mary's River	140
TRIBUTARY	< 1,311	SEDIMENTATION	1,000-1,600
MUNICIPAL	66	DEGRADATION	?
INDUSTRIAL	2	WATER-AIR TRANSFER	?
TOTAL	8,000-9,700	TOTAL	1,140-1,740

The vapour pressure of these molecules is then a critical property in terms of movement in the eco-system, particularly at the air/water interface and at the soil/water interface. We have yet to determine the transfer coefficients and fluxes at these interfaces in the gaseous phase, particularly the upward volatilization from soil and water. Verification will require the use of gas phase samplers.

For chlorinated dioxins and furans we know much less. Estimates place their vapour pressure in a range similar to that of PCB (16). They are expected to move in a relatively similar fashion (17). PCB in L. Huron biota show a slowly declining trend similar to L. Superior. Levels are sharply increased in Saginaw Bay

near Midland, Michigan and the outflows of the Tittibawassi and Saginaw Rivers. Increasing depositional rates were shown for total PCDD and PCDF in L. Huron. These increases correspond temporally with the production of chloro-aromatics and correspond spatially similar to PCBs with the proximity to Saginaw Bay and the Saginaw River system(1). The pattern of chlorinated dioxins and furans corresponded closely to that of combustion of chloro-aromatics and not fossil fuels such as coal. Sediment samples from the Great Lakes show this type of pattern save for L. Ontario where the pattern more closely fits that of pentachlorophenol. Distinct pattern changes for PCDD and PCDF were also evident in urban dust from various cities. Patterns of dust from Chicago are distinctly different from Midland, Michigan, which are different from Detroit.

Limited samples of soils near industrial incinerators at Midland, Michigan show elevated levels of PCDD ranging from ng/g to ug/g quantities, whereas rural areas show non-detectable levels. Urban areas near municipal incinerators in Chicago show detectable levels of PCDD but at levels ranging from pg/g to ng/g (18). Similar results were found in Hamilton, Ontario near a municipal waste incinerator (6).

The most recent and striking example of current discharge of waste contaminated with PCDD and PCDF is in the St. Clair River at Sarnia, Ontario, upstream of the drinking water supplies of the cities of Detroit, Michigan, and Windsor, Ontario (19). In this case a large, heavier than water mat of chemicals was found on the bottom of the Canadian side of the river in both 1984 and 1985. This material contained percentage levels of perchloroethylene, part per thousand levels of chlorobenzenes, hexachlorobutadiene, chloropropenes, octachlorostyrene, and PCBs and parts per billion levels of PCDD and PCDF. Although the octa- and hepta-chlorocongeners of PCDD and PCDF predominated, tetra-chlorodioxins characteristic of 2, 4 -D were found and mass spectral confirmation of 2, 4 -D was also obtained in the 1984 sample. It is important to realize that the presence of dioxin in the waste was the major public concern whereas it was the presence of water soluble compounds such as chloropropenes, chlorobenzenes, and perchloroethylene at extremely high concentrations which posed the greatest immediate threat to drinking water supplies. However, the less water soluble chlorinated dioxin/dibenzofuran molecules pose the greatest long-term threat in that they persist in sediment at ug/kg levels along approximately a 3 km stretch of the river bottom both upstream and downstream of the tarry mat of chemicals. Candidate sources of this discharge of chemicals, which are no longer manufactured by local industry, include an

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch007

industrial landfill, old sewer lines, and groundwater contaminated by previous deep well injection of similar wastes.

Humans In The Ecosystem

Herring Gulls and the coho salmon living in L. Ontario have a common food source, alewives and smelt which are the next step lower on the food chain. Alewives and smelt are the common source of PCB, DDE mirex, photomirex, and chlorobenzenes to the gulls and salmon (Table II)(20). The same relationship for 2,3,7,8-TCDD can be shown.(5)

Table II. Residues in Gulls and Fish in Ontario

	Alewives and Smelt (mg/kg)	Coho Salmon (mg/kg)	Herring Gull eggs (mg/kg)
PCB	2.21	5.7	138
DDE	.47	.97	17.4
Mirex	.09	.23	4.4
Photomirex	.03	.11	1.6
HCB	.024	.097	0.52
BHCH	.002	.012	.078
TCDD (5)	14×10^{-6}	30×10^{-6}	55×10^{-6}
Heptachlor epoxide	.003	.015	.007
Dieldrin	.029	.087	.060
	n=12	n=10 n=47	n=125

When one examines residues in the human population in Ontario (Table III), one finds exactly the same residues that are found in Herring Gulls and fish. Human body fat contains approximately the same concentrations as those found in alewives and smelt taken from L. Ontario.

Since Herring Gulls and coho salmon accumulate these residues from their common food supply, it seemed logical to consider the human exposure routes thoroughly, particularly the food supply. An early study by Bennet (23) followed total non-occupational environmental exposure of PCB to humans. This study showed that for PCB the atmospheric pathway contaminates plants directly, livestock indirectly, and ultimately contributes the major human environmental dosage of PCB. Exposure from drinking water and the atmosphere directly are inconsequential relative to food exposure. A more recent modelling effort (17) for 2,3,7,8-TCDD again shows food as the major route of

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch007

In Solving Hazardous Waste Problems; Exner, J.;
ACS Symposium Series; American Chemical Society: Washington, DC, 1987.

Table III. Residues in Humans in Ontario

	Adipose Tissue(21) (mg/kg)			Breast Milk(22) -fat basis (mg/kg)
	Kingston	Ottawa	Ontario	Ontario
PCB	3.0	2.0	2.1	0.756
DDT	3.429	2.694	4.069	0.983
Mirex	0.027	0.011	0.02	-
Photomirex	0.009	0.006	-	-
HCB	0.106	0.078	0.013	0.0184
BHCH	0.136	0.065	-	-
TCDD	12×10^{-6}	8.6×10^{-6}	-	-
Heptachlor epoxide	0.035	0.037	0.07	0.012
Dieldrin	0.036	0.043	0.10	0.194

exposure being transmitted from air to soil and then to plants and livestock. Based on these two studies a search of the literature was begun for residue values for PCDD and PCDF in food. No literature was available on general levels of dioxins in foods save for anomalous cases such as near chlorophenol production facilities and accidents.

In order to obtain exposure information on PCDD and PCDF as well as more realistic information on PCB and other persistent residues, a direct food composite survey was taken from supermarkets throughout the city of Toronto in the summer of 1985 (24). Only food grown in Ontario was sampled. Food was divided into five categories: leafy vegetables, fruit, root vegetables, milk, and meat and eggs. Chlorobenzenes were detected in all composites, with the most common congeners being the trichloroisomers. Lindane was detected in all composites except cows' milk. Other hexachlorocyclohexane isomers were also prevalent. DDT and its metabolites were still found in all composites. PCB were detected in fruit, milk and meat/eggs composites with the highest concentration found in fruit. Concentration of all the above residues, when detected, range from 0.1 to 1 ug/kg on a fresh weight basis.

Residues of PCDD and PCDF were detected in highest concentration in the fruit composite with the predominent residues being Penta-CDF, (0.28 ug/kg), Hex-CDF (2.0 ug/kg), Hex-CDD (0.45 ug/kg), Hep-CDF (6.6 ug/kg), Hep-CDD (1.2 ug/kg) Octa-CDF (1.2 ug/kg) and Octa-CDD (1.2 ug/kg). Residues in milk were the second highest with the predominant congeners being Tetra-CDF, Tetra-CDD, Pe-CDF, Hep-CDF, Hep-CDD, Octa-CDF, and Octa-CDD all found between 0.01 and 0.1 ug/kg. Root vegetables and the meat/eggs composite were contaminated at lower concentrations.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch007

These residue concentrations fit well with the fugacity III model prediction of Mackay et al (17) and begin to confirm that our food is the major exposure route for PCDD and PCDF.

Discussion

PCDD and PCDF are found widespread in the human population in a similar fashion to PCB, DDT and its metabolites, chlorobenzenes, and other common persistent organochlorines. Since food is influenced by environmental residue trends, human residue levels and exposure levels will likely follow environmental trends closely. In the Great Lakes basin area overall trends are not declining for either PCB or PCDD and PCDF except in localized areas.

Based on these findings several immediate recommendations are evident:

a) Risk estimates must consider the total multi media exposure routes for PCB, PCDD and PCDF. They must also consider that any additional risk posed by a potential increase in the exposure to these compounds must be considered from the point of view that exposure, and therefore risk, is already present.

b) Epidemiological studies must consider that populations with potential increased exposure are being compared with populations already exposed. Ambient exposure must be thoroughly documented and understood at current levels if we are to understand incremental risk.

c) Environmental records show that residue levels of these compounds increased during the period from 1940 to 1960 and no major downward shift has occurred on a widespread basis to match this increased deposition rate. Human residues for these compounds will not decline until measures are taken to either permanently contain or destroy the major sources of these compounds.

Sources

The major sources of PCDD and PCDF in the Great Lakes basin are low temperature municipal waste and sewage sludge incinerators, and leachate from past disposal practices, some of which were designed to contain wastes (6). All of these current source types are the result of engineering treatment designed to reduce the concentration of these compounds in discharges to water bodies.

In the case of sewage sludge incineration, chemical wastes have been preconcentrated in the sludge to reduce the aqueous effluents. Current low

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch007

temperature incineration practices lead to traces of PCDD and PCDF. Sludge absorption does little to reduce the net flow of persistent contaminants to the ecosystem but merely diverts them from the water to the atmosphere where they will either be transported or condensed out, ultimately entering the water. It is these municipal and sludge incineration sources that likely provide the greatest impact on human exposure through food. In the case of landfill leachate and areas where liquid wastes are available for cost-effective recovery, destruction would certainly seem the most desirable option over attempts to re-contain the waste.

The most cost-effective solution to both problems would be the on-site destruction of any available liquid waste containing persistent residues such as PCBs, PCDD and PCDF. This option would eliminate the transportation cost as well as risk in addition to removing any future risk, liabilty and cost. Remedial measures must not simply divert residue movement from the water into the air, such as in landfill remediation where temporary storage lagoons for leachate can allow rapid volatilization of PCBs, PCDD and PCDF.

Future Directions

1. Liability for possession and production of persistent toxic chemicals, particularly PCDD and PCDF is going to increase. In Ontario the recent promulgation of the "Spills Bill" has placed the liability for immediate cleanup and damages for a spill of toxic chemicals directly on the transporter and producer. This bill will eventually increase the cost of transportation of waste and promote on-site destruction devices.

2. The long-term liability for possession of persistent toxic chemicals, particularly PCDD and PCDF is also going to increase. Multi-media hazard, exposure and risk assessments are now becoming more common in the U.S.A. and Canada and will place a higher emphasis on direct and indirect toxic chemical exposure from the atmosphere. Storage, transfer, waste reduction and destruction facilities, and even some recycling facilities will come under severe pressure to meet new atmospheric standards. These standards will include provision for contemporary local air transport models which are key to the assessment of exposure and risk posed by highly toxic chemicals. Effective destruction will remove this liability.

3. Over the next decade aquatic effluents as well as atmospheric emissions in the Great Lakes basin will likely be considered in terms of loading

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch007

requirements rather than current concentration guidelines. This approach is already being used by the six states in co-operation with the USEPA who are grappling with a loading reduction strategy for PCB into Lake Michigan. This same approach is called for by the Canadian and U.S. technical committees on the Niagara River and a new IJC Loadings Task force is beginning work on L. Ontario. If loadings in these areas are to be reduced by 50 %, destruction of in situ sources of chlorinated persistent compounds such as PCB, PCDD and PCDF will most likely be required.

4. Finally, PCDD and PCDF are becoming recognized as one of the most prevalent toxic chemical groups in industrialized areas. They are prevalent in human tissues. Preliminary evidence verifies exposure models showing food as the major vector. Dioxins may be one of the most prevalent residues in certain food types such as milk and fruit. Since emissions to the air are the most likely pathway to food, our priority must lie with reducing the creation of these compounds through improper combustion of organochlorine precursors and reducing the overall cycling of these compounds by destruction of existing major sources to the air, water and soil of our ecosystem.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch007

Literature Cited

1 Czuczwa, J.M. and Hites, R.A. 1986. Environ. Sci. Technol. 1986. 20 No. 2 195-200.
2 Swain, W.R. Abstracts 25th Conf. International Association for Great Lakes Research. 1982.
3 Eisenreich, S.J., B.B. Looney, and J.D. Thornton. Environ. Sci. Technol. 1981. 15: 30-38.
4 Hallett, D.J., Hutter, M.P. In "Environmental Fate of Chlorinated Dioxins and Related Compounds"; O. Hutzinger, Pergamon Press, 1982.
5 National Research Council of Canada, Ottawa, Canada. 1982. NRCC Publication No. 18574, p.251.
6 Ontario Ministry of the Environment. Scientific Criteria Document No. 4-84. 1985. Toronto, Canada.
7 Hallett, D.J. In "Persistent Toxic Substances and the Health of Aquatic Communities." John Wiley and Sons In press.
8 Durham, R.W. and Oliver, B.G. J. Great Lakes Res., 1983. 9:160-168.
9 Hallett, D.J., and Brooksbank, M.G. Chemosphere 1986. (in press).
10 Organization for Economic Co-operation and Development. Proc. PCB Seminar the Netherlands. 1983. p. 410.
11 Rappe, C. In "Solving Hazardous Waste Problems: Remedial Action at Sites Contaminated by

Polychlorinated Dioxins" 191st ACS National Meeting. New York, N.Y. 1986. p.86.
12 Mackay, D. and Paterson, S. Environ. Sci. Technol. 1982. 16 6554A.
13 Mackay, D. and Paterson, S. In "Environmental Exposure from Chemicals" Neely, B Ed. CRC Press (in press).
14 Bidleman, T.F. and Christensen, E.J. J. Geophys. Res. 1979 84 No. C12 7857-7862.
15 Murphy, T.J. and Rzeszutko, C.P. J. Great Lakes Res. 1977 3, 305-312.
16 Rordorf, B.F. Chemosphere, 1985, 14, No6/7 885-892
17 Mackay, D. Paterson, S. and Cheung, B. Chemosphere, 1985, 14 No 6/7 859-864.
18 Dow Chemical. "The trace chemistries of fire." 1978. 46pp.
19 "St. Clair River Pollution Investigation." Ontario Ministry of the Environment. 1985.
20 Norstrom, R.J., Hallett, D.J., and Sonstegard, R.A. J. Fish. Res. Board. Can. 1978. 35 1401-1409.
21 Williams, D.T., LeBel, G.L., and Junkins, E. J. Toxicol. and Environ. Health. 1984, 13:19-29
22 Frank, R., Ontario Ministry of Agriculture and Food, (personal communication).
23 Bennet, B.G., Sci. Total Environ., 1983. 29:101-111.
24 Davies, D. 1986. Proc. World Conf. on Large Lakes., Mackinac Mich.

RECEIVED January 14, 1987

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch007

Chapter 8

Experimental and Calculated Physical Constants for 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin

Leland Marple, Robert Brunck, Bernard Berridge, and Lewis Throop

Syntex Analytical and Environmental Research, Syntex (USA), Inc., 3401 Hillview Avenue, Palo Alto, CA 94303

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch008

The measurement of water solubility, water-octanol and water-soil partition coefficients presented several unique challenges. Novel experimental methods and data are reported for these physical constants. In some cases, experimental data is at odds with earlier published values. Comprehensive comparison of all available estimated, calculated, and experimental data are presented. The new values impact the calculation of the mobility of dioxin in soil, as well as other distribution properties.

In spite of many studies on the physical properties of dioxin, we were unable to find any definitive work that measured the water solubility, octanol-water partition, or soil-water partition constants or coefficients for 2,3,7,8-tetrachlorodibenzo-p-dioxin (dioxin). The importance of these values in predicting the migration of dioxin in an environmental setting led us to undertake a program to measure these physical constants.

WATER SOLUBILITY

Water solubility values in current use that ranged from 0.2 to 2 ppb were at odds with our observation that in the absence of suspended matter, the amount of dioxin in environmental water samples analyzed in this laboratory was rarely more than 2-3 parts per trillion (ppt). We anticipated that our main difficulties in measuring dioxin solubility would be separation of solid, adsorbed, and dissolved dioxin, and the measurement of soluble dioxin in the equilibrated solutions. During the last two years, we developed three techniques for measuring the water solubility of dioxin. The merits and results of each of the methods and comparison with solubility values from the literature are given below.

Our first method began by subliming 14C-radiolabeled dioxin onto the cold finger of a small sublimation flask. The sublimate was

0097-6156/87/0338-0105$06.00/0
© 1987 American Chemical Society

then transferred to a second flask that had been silanized and filled with distilled water. After equilibration, the major portion of the water was removed, the dioxin was extracted with hexane, the hexane extract was evaporated to near dryness, and the residue was taken up in Oxifluor scintillation fluid for counting.

Measurement of the specific activity of the 14-C dioxin by GC/EC allowed calculation of the amount of dioxin that was dissolved. Since the amount of dioxin that dissolved was so small compared to the amount on the cold finger, the same sublimate could be used for all equilibrations. The results for the solubility of 14-C dioxin are given in Table I.

Table I. Solubility of 14-C Dioxin in Water from a Sublimed Film

Equil. time days	Conc. TCDD ppt	Solubility moles/liter
1	6.8	2.0×10^{-11}
3	12.7	3.9×10^{-11}
5	12.3	3.85×10^{-11}

These values were much lower than the 200 ppt value reported by Crummett and Stehl (1).

To avoid concerns about the radiochemical purity of the labeled dioxin, the solubility of unlabeled, 98% pure (Cambridge Isotope Labs) dioxin was determined by a scaled up version of the method just described. In this experiment, the entire contents of the flask was transferred to a separatory funnel, 13-C dioxin was added as an internal standard, and the combined dioxins were extracted with hexane. A capillary GC/MS method was used for quantitation (5). A summary of results is given in Table II.

Table II. Solubility of Unlabeled Dioxin in Water From a Sublimed Film

Equil. time days	Conc. TCDD ppt	Solubility moles/liter
1	18.6	5.8×10^{-11}
3	15.4	4.8×10^{-11}
5	25.1	7.8×10^{-11}
5	20.7	6.4×10^{-11}
5	16.9	5.2×10^{-11}

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch008

The high surface to volume ratio of dioxin film afforded by the sublimation flask allows equilibrium to be reached in a short time, and allows easy, complete separation of the solid dioxin from the equilibrated solution. The values obtained were consistent with those obtained with radiolabeled dioxin.

The third method involved equilibration of dioxin present in a compact film on a glass slide, (formed by evaporation of dioxin dissolved in hexane), with water in a 2 liter volumetric flask. A glass stirrer was used for mixing the contents. To avoid problems with the formation of an interfacial film, the flask was rigorously cleaned with boiling concentrated nitric acid, and the cleaned flask was filled to the neck with water for equilibration. A capillary GC/MS method was used for dioxin analysis. Solubility results using this method are given in Table III.

Table III. Solubility of Unlabeled Dioxin in Water from a Compact Film

Equil. Time days	conc. TCDD ppt	Solubility moles/liter
3	10.0	$3.1x\ 10^{-11}$
3	9.1	2.8×10^{-11}
3	12.2	3.8×10^{-11}

A summary of all experimental values (mean values), and earlier reported values is given in Table IV.

Table IV. Summary of Values for the Water Solubility of Dioxin

Solubility, ppt	Method	Source
2000	?	Plimmer, et al (2)
600	?	Kearney, et al (3)
200	?	Crummet, Stehl (1)
12.5	14-C film	Marple, et al (4)
19.3	thin film	Marple, et al (4)
10.4	compact film	This work
8.0	?	Overcash (5)

The fact that our three methods give very nearly the same solubility value is taken as evidence that we are measuring the water solubility free of artifacts arising from adsorption, particle suspension, and other phenomena.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch008

OCTANOL/WATER PARTITION

The partition between octanol and water is another useful physical property for which we could find no experimental data in the literature. While we anticipated several experimental problems in the measurement, we soon realized that the primary problem was the physical separation of equilibrated phases. An equilibration cell was designed, Figure 1, in which diffusion was the main mechanism of mass transfer of dioxin from the octanol to the water phase. A slight mechanical movement of the water phase hastened the distribution of dioxin within that phase. Although the volume of the side arm was small in comparison to the volume sampled, the liquid in the standpipe was periodically flushed back into the equilibrating chamber by application of a slight positive pressure to the top of the standpipe.

Equilibrations were started using both mutually unsaturated and mutually saturated phases. The results (Table V) show that equilibrium is achieved quickly starting from mutually saturated phases.

Table V. Variation of Octanol-Water Partition Coefficients for Dioxin with Equilibration Time

Equil. Time	Log K ow	
days	Phases Unsaturated	Phases Saturated
5	4.80	
5		6.53
12	5.70	
15		6.95
20	6.52	
31	6.22	6.59

The increase in Kow for unsaturated phases reflects a decreasing dioxin content with time in the water phase. This is thought to be due to the transfer of octanol solvated dioxin into the water phase at the start of the equilibration while the two phases are in the process of reaching mutual saturation. The octanol solvate would be unstable in the aqueous phase, however, preferring to dissociate into octanol and water solvated dioxin. A mean value of the partition coefficient was calculated from measurements in which the systems were thought to be at equilibrium. Table VI compares our mean experimental value with other values found in the literature.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch008

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch008

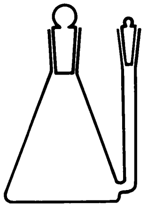


Figure 1. Equilibration cell used for Octanol-water partition.

Table VI. Summary of log Kow Values for Dioxin

Log Kow	Calc from water	
6.9	calc.	Johnson (6)
7.14	calc.	Perkaw, et al (7)
8.93	calc.	Webster, et al (8)
6.15	?	Kenaga (9)
6.64	slow diffusion	Marple, et al (13)
6.40-6.59	calc from water solubility	Tables I-III

It should be noted that the octanol-water partition coefficient calculated from the Law of Distribution Between Phases and the experimental water solubility agrees well with our experimetally determined partition coefficient. This result is expected since the molar solubility of dioxin in both octanol and water is sufficiently low at saturation that there is no significant impact on the activity coefficient of dioxin in either phase. Further, solubilities of octanol in water, and water in octanol are so slight that there is no significant difference between dioxin solubilities for the pure solvents compared to mutually saturated solvents.

SOIL/WATER PARTITION

The partition of dioxin between soil and water was expected to be largely dependent upon the organic carbon content of the soil. Experimentally measured partition coefficients are normalized for organic carbon by dividing by the organic carbon content. Although there are a number of methods for the measurement of organic carbon, the Walkley-Black method (11) is most frequently used. Using our experimental solubility values and the Koc-solubility relation published by Chiou, Peters, and Freed (12) shown in Table VII, we estimated that log Koc would be in the vicinity of 6.39 - 6.54.

Table VII. Koc-Solubility Regression Equations

Chiou, Peters, Freed (11)

$$\text{Log Koc} = 4.040 \ (\pm 0.038) - 0.557 \ (\pm 0.012) \ \text{Log S}$$

S = solubility in micromoles/liter, n = 15

Kenaga, Goring (12)

$$\text{Log Koc} = 3.64 - 0.55 \ \text{Log S}$$

S = solubility in parts per million

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch008

If instead the empirical relation developed by Kenaga and Goring was used (13), the log Koc estimate ranged from 6.24 to 6.49.

The main experimental problem was unexplained loss of dioxin to the equilibrating system. In many of our preliminary experiments, small amounts of dioxin equilibrating with water and soil simply disappeared. Uncontrolled adsorption on the surface of the equilibrating vessel, most likely as a scum, was thought to be responsible for the loss. When the surface of all glass in contact with the water phase was cleaned with hot, concentrated nitric acid, the loss was small enough to permit measurement of the dioxin levels in both soil and water phases.

Two representative Missouri soils were selected for investigation: a red clay with a 0.3% organic carbon content, and an alluvial stream soil with a 1.3% organic carbon content. The soils were air dried and screened to remove rocks and gravel before use. Dioxin was added to the system in two ways: a solution of dioxin in toluene was evenly deposited on the soil, and the soil dried before use; or, dioxin was deposited as a visible film on a glass plate which was put into the equilibrating system. A two liter borosilicate flask and a mechanical glass stirrer sealed at the neck were used for equilibration. The apparatus was protected from the light to avoid photodecomposition. Upon completion of the equilibration, the suspension was allowed to settle overnight. A sample of the clear supernatant was drawn off with pipette, then the bulk of the water phase was drawn off to allow collection of the soil. After adding 13-C dioxin to each sample as an internal standard, the dioxins were isolated from each sample by solvent extraction and adsorption chromatography. A capillary GC/MS method was used for the analysis of dioxins in both phases.

Results of our soil partition measurements are collected in Table VIII.

Table VIII. Summary of Organic Carbon Soil-Water Partition coefficients for Selected Missouri Soils

Soil Type	Equilibration Time	Log Koc
Red Clay	25	6.45
	112	6.54
	72	6.22
	25	6.39
Stream Alluvial	29	5.96
	20	6.09

The first two entries for clay soils are the result of starting with dioxin adsorbed on soil, and the following two are the result of suspending dioxin in the equilibrating system. Although the conversion to logarithms appears to reduce the variation of the measurements, the range of values is quite narrow considering the potential variability of the soil specimens.

The reproducibility of the experiment was briefly examined in two equilibrations where we measured the amount of dioxin transfered to the water phase in contact with a constant amount of dioxin-soil

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch008

In Solving Hazardous Waste Problems; Exner, J.;
ACS Symposium Series; American Chemical Society: Washington, DC, 1987.

mixture over a period of several days. Although we expected the dioxin level in solution to increase with time, there was an equal and parallel decrease in the dioxin content after the first sampling period. When both soil and water phases were analyzed at the end of the experiment, it was clear that about 40% of the dioxin had migrated to a fixed interface within the equilibrating system.

In addition to the Koc-Solubility relations already mentioned, we estimated Koc from the Koc-Kow relation developed by Karickhoff et. al., Log Koc = 1.00 log Kow - 0.21 (15). Our estimate and earlier reported values using this relation are compared in Table IX.

Table IX. Organic Carbon Soil-Water Partition Coefficients for 2,3,7,8-TCDD Calculated from Karickhoffs Regression Equation and Octanol-Water Partition Coefficients

Log Koc	Source
6.90	Marple (15)
6.51	Mabey, et. al (16)
6.43	Marple (this work)

Experimental values of Koc appear to be in excellent agreement with Koc estimated from experimental Kow value. Recalling that log Koc estimates from solubility relations were 6.39 - 5.64 and 6.24 - 6.49, it is clear that our experimenatl values for the water solubility, octanol-water partition coefficient, and soil organic carbon-water partition coefficient are internally consistent. We attribute this to having taken steps to minimize losses to unknown parts of the equilibrating system, and to correct for losses by the analysis of dioxin in each of the phases involed in the partition system.

Literature Cited

(1) Crummett, W.B. and Stehl, R.H., Environmental Health Perspect. 1973, 5, 15.

(2) Plimmer, J., Klingbiel V., Crosby, D., Wong, A., "Chlorodioxins - Origin and Fate", Adv. Chem. Series 120, E.H. Blair, Ed. p. 46.

(3) Kearney, P., Isensee, A., Helling, C., Woolson, E. Plimmer, J. loc cit. p. 106.

(4) Marple, L.W., Brunck, R., Throop, L., Environ. Sci. Technol., (accepted for publication.).

(5) Overcash, M.R., Private Communication, N. Carolina State Univ., Raleigh, No. Carolina.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch008

(6) Johnson, H., "Aquatic Fate Process Data For Organic Priority Pollutants", W.R. Mabey, ed. EPA Final Report, Contract 68-01-3867.

(7) Perkaw, J., Eschenroeder, A., Goyer, M., Stevens, J., and Wechsler, A., "An Exposure and Risk Assessment for 2,3,7,8-Tetrachlorodibenzo-p-dioxin", Office of Water Regulations and Standards, US EPA, Wash. D.C. (1980).

(8) Webster, Sarna, L. Muir, D. Publication pre-print, "Kow of 1,3,6,8-TCDD and OCDD by Reverse Phase HPLC", Univ. of Manitoba, Winnipeg, Manitoba, Canada R3T 2N2.

(9) Kenaga, E., Environ. Sci. Technol. 1980, 14, 553.

(10) Walkley, A., Black, I.A., Soil Sci, 1934, 37, 29-38.

(11) Chiou, C.T., Peters, L.J., Freed, U.H., Science, 1979, 206, 831-832.

(12) Kenaga, E.E., Goring, C.A.I., "Relationship Between Water Solubility Soil-sorption, Octanol-water Partitioning and Bioconcentration of Chemicals in Biota, ASTM Third Aquatic Toxicology Symposium, Oct. 17-18, New Orleans, LA.

(13) Marple, L.W., Berridge, B., and Throop, L. "Measurement of the Water-Octanol Partition Coefficient of 2,3,7,8-tetrachlordibenzo-p-dioxin", Environ, Sci. Technol. 1986, 20, 397-399.

(14) Karickhoff, S.W. Brown, D.S. Scott, J.A., Water Research 1979, 13, 241.

(15) Mill, T., "Prediction of the Environmental Fate of Tetrachlorodibenzo-p-dioxin", presented at the Conference on Dioxins in the Environment, Mich. State Univ., Mich. December, 1983.

(16) Mabey, W.R., Smith, J.H., Podoll, R.T., Johnson, H.L., Mill, T., Chou, T.W., Gates, J., Partridge, I.W., Jaber, H., Vanderberg, G., "Aquatic Fate Process Data by Organic Priority Pollutants: 1982 Final Report, US EPA, Wash. D.C. 20460.

RECEIVED December 2, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch008

Chapter 9

Experiments on the Mobility of 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin at Times Beach, Missouri

Raymond A. Freeman, Frederick D. Hileman, Roy W. Noble, and Jerry M. Schroy

Monsanto Company, St. Louis, MO 63166

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch009

The Times Beach Dioxin Research Station was constructed in 1984 under the direction of the University of Missouri and the Missouri Department of Natural Resources. The station offers investigators the opportunity to conduct experiments under field conditions on a well characterized soil. During the Summer of 1984, a series of six experiments were established by Monsanto at the station to study the environmental transport of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) under field conditions. The plots initially contained uniformly sized soil that was thoroughly mixed to provide a consistent TCDD level. The experiments were designed to study the movement of TCDD in soil. The experiments found that:

1. Vaporization and/or photodegradation at the surface of a contaminated soil is a loss mechanism for TCDD.
2. The presence of water and/or sunlight is an important environmental parameter for the mobility of TCDD. Water appears to weaken the binding of TCDD to the soil matrix.
3. TCDD accumulation by plants has a minimal impact on the soil concentration of TCDD.

The environmental behavior of chemicals with a very low volatility, such as 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), has been the subject of several studies over the last few years. The focus of these studies has varied from the development of physical property data (1,2) to lab and field tests of the mobility and/or fate of TCDD. The mobility and fate studies have involved research on the leaching of dioxin from the soil (3), vaporization of the organic contaminants into and out of the soil (4), the biodegradation or chemical decomposition of the organics in the soil column (5), and the photodegradation of the organics in the soil (6,7). These studies usually have involved laboratory investigations or computer simulation of tests started many years ago.

0097-6156/87/0338-0114$06.00/0
© 1987 American Chemical Society

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch009

When the State of Missouri established the Times Beach Dioxin Research Station in 1984 (8), it provided, for the first time, a facility where researchers could run relatively large field tests using aged TCDD contaminated soil. Previous studies (4) suggested that the mobility of TCDD in the soil was very slow and depends strongly on the ambient meteorological conditions. The Times Beach Dioxin Research Station offered the type of tight security needed for the long term testing required to establish the mobility characteristics of TCDD. The test station also offered a site where the initial soil contaminant level could be analyzed directly at the start of the experiments.

During the Summer of 1984, a series of six experiments were established by Monsanto at the station to determine the rate of environmental transport of (TCDD) under field conditions. The plots initially contained uniformly sized soil that was thoroughly mixed to provide a consistent TCDD level. The experiments were designed to study the movement of TCDD in a soil:

1. saturated with water (all pores filled with water),
2. maintained in a "damp" but unsaturated condition,
3. under natural field conditions,
4. covered with approximately a 5 cm layer of TCDD free soil,
5. maintained in a dry environment and protected from the wind, and
6. maintained in a dry environment, isolated from the sun with an impermeable metallic insulation cover, and protected from the wind.

At various times, soil core samples were taken, carefully sectioned, and analyzed for TCDD.

Experimental Design

The overall goal of this research was to study the mobility of TCDD under actual field conditions to determine the influence of such natural phenomena as: rain, floods, sunlight, and heat. The Missouri Department of Natural Resources established a Dioxin Research Station at the Times Beach Site during the late Spring and early Summer of 1984. Two blocks of Laurel Road were used to establish the Research Station. The TCDD contaminated soil was removed from the road and stainless steel bins (183 cm wide, 244 cm long, 61 cm deep) were buried in the roadbed. The contaminated soil, previously removed from the road, was homogenized by mixing, screened to remove large rocks, and placed into the stainless steel bins. The soil composition has been reported as 11.4 percent sand, 55.2 percent silt, 33.4 percent clay, and 2.0 percent organic matter (9). The process of preparing each bin was monitored to insure uniform mixing and TCDD levels. After preparation, each bin was sampled and covered with an impermeable metallic insulation cover. For more details on the construction procedures used see Yanders (8).

Monsanto rented four bins at the Times Beach Dioxin Research Station (TBDRS) during the summer of 1984. Six complementary experiments were installed in the four bins as:

1. PLOT A - TBDRS Bin 4B
   A timer controlled watering system was installed to keep the bin saturated with water (all pores filled with water).
2. PLOT B - TBDRS Bin 4C
   A timer controlled watering system was installed to keep the bin damp but unsaturated with water.
3. PLOT C CONTROL - TBDRS Bin 5A
   The bin was partitioned into two equal areas (east and west) using an aluminum divider. The east side of the bin was maintained under natural field conditions as a field control. The west side of the bin was used as described in point 4 below. See Figure 1 for a plan of bin 5A.
4. PLOT C NURSERY - TBDRS Bin 5A
   The bin was partitioned into two equal areas (east and west) using an aluminum divider. A total of 9,988 g of 3 mm glass beads was evenly applied to the west side (area = 2,297 cm2) of the bin to serve as a marker layer. The west side was then covered with approximately a 5 cm layer of TCDD free nursery soil (<1 ppt of TCDD). This clean soil over dirty soil experiment was designed to detect any upward TCDD movement. The east side of the bin was used as described in point 3 above.
5. PLOT D UNSHADED - TBDRS Bin 5B
   The bin was covered with a plastic sheet, permeable to ultraviolet radiation, to keep out rain and maintain a dry environment and protect it from the wind. The east side of the bin was allowed to remain in this state and the west side of the bin was used as described in point 6 below. See Figure 2 for a plan of bin 5B.
6. PLOT D SHADED - TBDRS Bin 5B
   One-half of the soil contained in the bin, discussed in point 5 above, was covered with an impermeable metallic insulation cover (west side of bin was covered). This serves as the basic control for the field study. The soil in this half of Bin 5B was protected from rain, wind, and direct sunlight. The soil concentration profile of TCDD, in this half of the bin, was not expected to change with time. The east side of the bin was used as described in point 5 above.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch009

Soil Sample Preparation

Soil and clay samples were dried, weighed and spiked with 24.0 ng of ^{13}C labeled 2,3,7,8-TCDD. Ten g of washed anhydrous sodium sulfate were added and the samples were stirred, allowed to stand for 12 hours and stirred again. Samples were mixed again immediately before the addition of extraction solvents. The extraction process involved the addition of 15 mL of methanol (all solvents are Burdick and Jackson "distilled in glass" grade), stirring, and the subsequent addition of 150 mL of hexane. The samples were shaken for three hr on a wrist action shaker and the extraction solvent was decanted from the soil. The solvent was then successively extracted with 50 mL of water, concentrated sulfuric acid (until clear), water, and 20 percent potassium hydroxide and water. The hexane was dried with 10 g of anhydrous sodium sulfate and passed through a mixed phase column. The column was prepared from a 25 mL disposable pipet containing (top to bottom) 2 g of silica gel, 4 g of silica gel plus 40 percent sul-

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch009

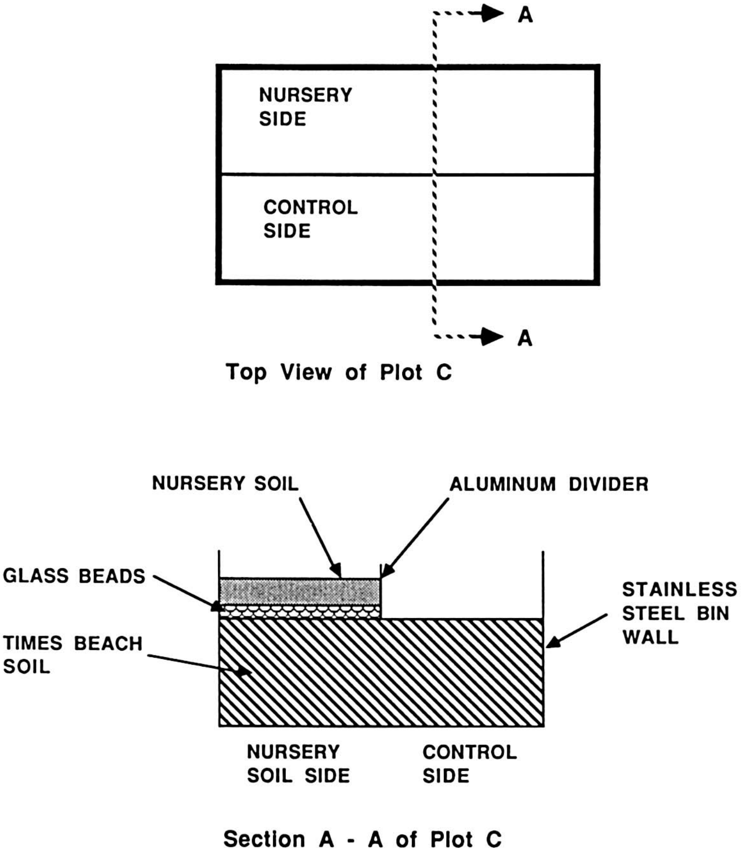


Figure 1. Diagrammatic Plan for Plot C

In Solving Hazardous Waste Problems; Exner, J.;
ACS Symposium Series; American Chemical Society: Washington, DC, 1987.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch009

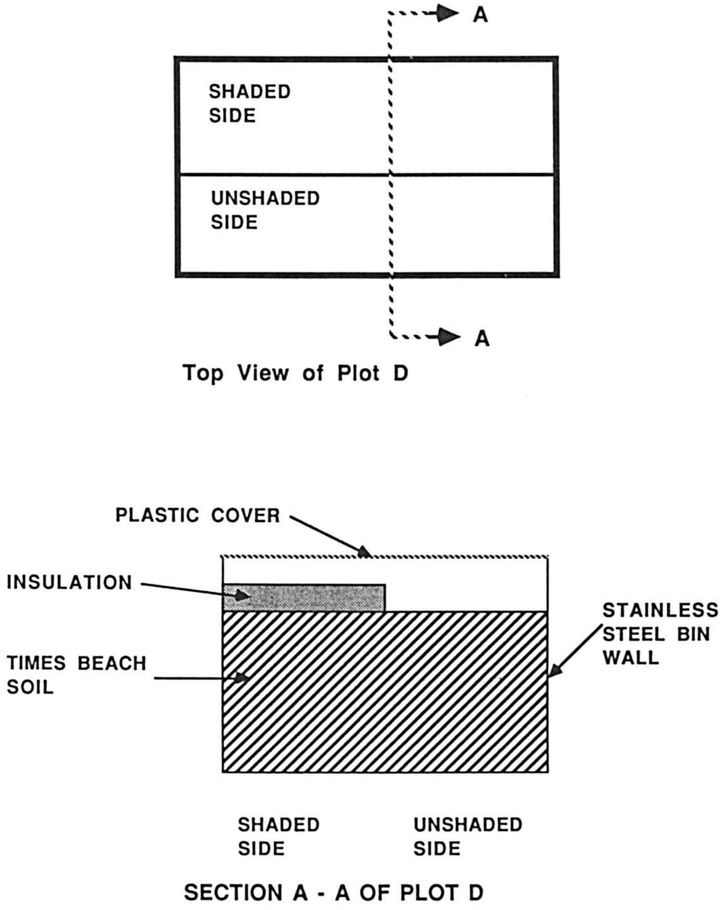


Figure 2. Diagrammatic Plan for Plot D

furic acid, 1 g of silica gel, 2 g of silica gel plus 36 percent potassium hydroxide, and 1 g of silica gel. The column was eluted with an additional 50 mL of hexane. All the eluant from the mixed phase column was passed directly onto a column prepared from a 10 mL disposable pipet containing 2.5 g of Woelm basic alumina. The alumina column was first eluted with 10 mL of 2 percent methylene chloride/hexane (v/v) and this fraction was discarded. An additional elution was made with 15 mL of a 50 percent methylene chloride/hexane (v/v) solution to elute the TCDD off the column. To this fraction was added a dodecane keeper. The sample was then concentrated to either 30 uL or 50 uL under a gentle stream of nitrogen and submitted for GC/MS analysis.

Plant Sample Preparation

Five-g samples of plant matter (grass and/or plants) were weighed into glass bottles. When weeds were encountered, large stems were avoided. The plants in each bottle were spiked with 100 uL of 100 pg/uL ^{37}Cl-TCDD in iso-octane by sprinkling the solution over the plant matter and allowing the solvent to evaporate. The plants were then washed three times by shaking with 50 mL portions of hexane for 15 min. The hexane rinses were pooled for each sample and spiked with 20 uL of 240 pg/uL ^{13}C-TCDD. Blank grass samples were run in the same manner. The hexane rinse samples were then taken through the cleanup procedure described below.

After the plant matter was rinsed with hexane as described above, the plants were spiked with 20 uL of 240 pg/uL ^{13}C-TCDD. Each sample was digested in 50 mL of 50 percent (v/v) H_2SO_4 and shaken for 15 min. The resulting solution was extracted three times with 50 mL portions of hexane. The hexane was removed from the H_2SO_4 sample solution by centrifuging at 3000 RPM for 30 to 60 minutes after each hexane extraction. The hexane extractions were then pooled and taken through the cleanup procedure described below.

The hexane solutions described above were subjected to the same cleanup procedure used for the soil samples. The samples were then concentrated to 10 uL under a gentle stream of nitrogen and submitted for GC/MS analysis.

Sample Analysis

The GC/MS analyses were conducted on a Hewlett Packard 5987 GC/MS instrument. The gas chromatographic separations used either a 60 m SP-2330 capillary column or a 30 m DB-5 capillary column. The mass spectrometer was operated in the selected ion monitoring mode analyzing for ions characteristic of the native (unlabeled) 2,3,7,8-TCDD (m/z 320, 322), carbon-13 labeled 2,3,7,8-TCDD (m/z 332, 334). Quantification was achieved by ratioing the area of the native 2,3,7,8-TCDD to the area of the carbon-13 labeled 2,3,7,8-TCDD. The ratio was compared to that obtained for a set of analytical standards and the amount of 2,3,7,8-TCDD determined. The levels present in the original soil or plant sample were determined by dividing the weight of the sample into the measured weight of 2,3,7,8-TCDD present.

For the plant samples an additional analysis was carried out for the distribution of the ^{37}Cl-TCDD between the rinse and the digested sample. Since the ^{37}Cl-TCDD was originally spiked onto the outer

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch009

surface of the plants, this analysis allowed for the determination of the effectiveness of the rinsing procedure. For all plant samples, less than 2 percent of the spiked ^{37}Cl-TCDD remained on the plants after the rinse procedure was completed.

Chronology of Field Samples Taken

On August 7, 1984 the experiments in plots A, B, and D were established. Two soil core samples were taken from plots A, B, C, and D on August 8, 1984. On August 24, 1984 the experiments in plot C were established. The nursery dirt used in this experiment was purchased from a local nursery and contained no TCDD at a detection limit of 1 ppt by weight. On October 9, 1984 a soil core was taken from Plot C - Nursery side. The cores taken on August 8, 1984 were segmented into approximately 2.5 cm (1 inch) segments and were placed into glass bottles. The bottles containing the 2.5 cm segments were stored in darkness until analysis during the first quarter of 1986. The core taken on October 9, 1984 was refrigerated in darkness until being segmented into 1 cm increments for analysis during the first quarter of 1986.

On July 9, 1985, one soil core was taken from plots A, B, C - control, C - Nursery, D - shaded, and D - unshaded. In addition, plants were growing on plots A, B, C - control, and C - nursery. The plants were cut using garden shears at the soil surface. Care was taken not to disturb the soil. Any plant matter falling on soil surface was assumed to be contaminated and was not used in the grab samples taken for TCDD analysis. The total weight of plant matter cut was measured in the onsite soil laboratory. Samples of the plant matter were taken and placed into standard glass sample jars. These samples were returned to the Monsanto Laboratory for refrigerated storage awaiting analysis. The six soil cores taken on July 9, 1985 were refrigerated in darkness until being segmented into 1 cm increments for analysis during the first quarter of 1986. To reduce the amount of plants growing on the experimental plots, a total of 1.004 g of the herbicide Oust was applied to plots A, B, C - control, and C - Nursery. A sample of Oust was found to be free of TCDD at a detection limit of 20 ppt by weight.

On November 4, 1985, one soil core was taken from plots A, B, C - control, C - Nursery, D - shaded, and D - unshaded. These cores were returned to the Monsanto Laboratory for refrigerated storage while awaiting analysis. The soil cores were segmented into 1 cm increments and were analyzed for TCDD during the first quarter of 1986. In addition, soil surface samples were obtained by scraping the surface of Plots A, C - Control, and D -Shaded with disposable stainless steel spatulas. The depth of the soil surface collected in this manner was approximately 3 mm. These surface samples were returned to the Monsanto Laboratory for storage and later analysis. A small number of plants was found to be growing on plots A, B, C - control, and C - Nursery. The plants were cut at the surface using garden shears and placed into plastic sample bags. All of the plant matter which did not fall onto the soil surface was returned to the Monsanto Laboratory for refrigerated storage while awaiting analysis. It is estimated that at least one-half of the weight of growing plants were collected in this manner. After the application of the herbicide Oust, very few plants grew on the plots.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch009

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch009

Soil Study Results

All soil cores were segmented into either 2.54 cm (1 inch) or 1 cm increments and analyzed for TCDD. The TCDD concentration data are plotted in Figures 3 thru 7 for Plots A, B, C - Control, D - Shaded, and D - Unshaded. Since the experiment in Plot C - Nursery was designed to yield only a yes or no answer on the mobility of TCDD, it is discussed separately below. Each concentration point is plotted at the midpoint of the corresponding soil increment depth. For example, the measured concentration for the 8 cm to 9 cm increment would be plotted at a depth of 8.5 cm. These experiments were designed to detect the loss of TCDD from the surface soil layers. Thus, the line shown as the mean in each figure is the average of all measured TCDD concentrations, excluding the surface layer of 0 to 1 cm or 0 cm to 2.54 cm. The dashed lines above and below the mean represent the 2 standard deviation limits about the mean. Any point falling outside the upper and lower limits are suspected to be statistically different from those inside the limits. As can be seen from Figures 3 thru 7, the data are very consistent.

In Figure 3, there is a suspect point plotted at 6.35 cm, taken in August 9, 1984, with a concentration of 8.2 ppb. There were two cores taken in August of 1984 for Plot A. The corresponding concentration in the second core was found to be 98 ppb. All TCDD analyses for the August 1984 cores yield concentrations between 86 and 115 ppb. We conclude that sample is an outlier probably due to human error in the analysis.

Not plotted on Figure 6 is a suspect point of 326 ppb in the 4 cm to 5 cm soil increment for Plot D - Shaded taken on November 4, 1985. The concentration in the 3 cm to 4 cm soil increment is 175 ppb. The concentration in the 5 cm to 6 cm soil increment is 174 ppb. The 4 cm to 5 cm soil increment was found to contain 134 ppb when sampled in July of 1985. In addition, the 4 cm to 5 cm soil increment for the other side of Plot D (Unshaded side) was found to be 186 ppb in the November 1985 core. There is no known mechanism for this kind of movement of TCDD in a protected dry environment. Thus, we conclude that the sample is an outlier probably due to human error during analysis.

The two outlier samples were not used during later data analysis. All other points except the soil surface samples are consistent with normal statistical scatter in measured data.

If vaporization and/or photodegradation of TCDD at the soil surface occurs, the soil near the surface should have lower TCDD concentrations than the soil layers below the surface. Previous calculations by the authors indicated that the the lowering would only be detectable in the top 1 cm of soil after a normal hot Missouri Summer.

Surface soil scrapings were taken from Plots A, C - Control, and D - Shaded on November 4, 1985. The results of TCDD analyses of these surface samples were 33 ppb, 66 ppb, and 121 ppb for Plots A, C - Control, and D - Shaded respectively. These surface TCDD concentrations are compared to the concentrations in the subsurface soil layers in Figures 3, 5, and 6. Since it is impossible to take a sample of zero thickness, the surface scraping data are plotted at a depth of 0.15 cm.

For plots exposed to water and sunlight (Plots A and C - Control) the surface scrapping TCDD concentrations are significantly

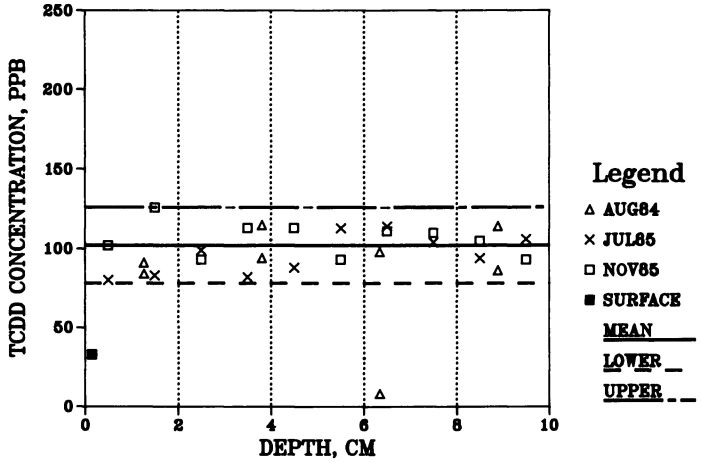


Figure 3. TCDD Concentration Profile for Saturated Conditions - Plot A

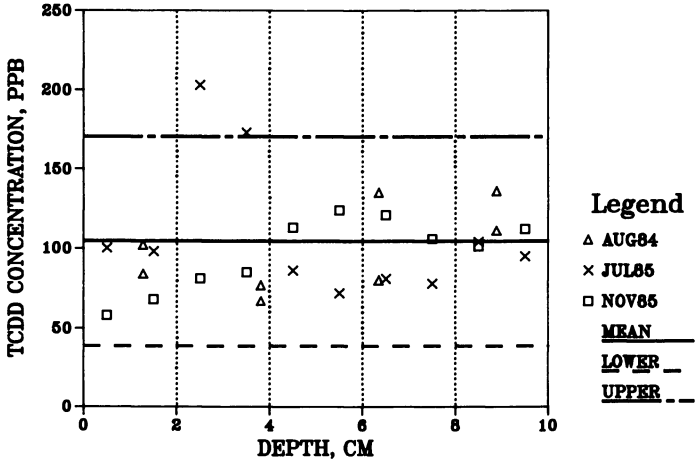


Figure 4. TCDD Concentration Profile for Damp Conditions - Plot B

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch009

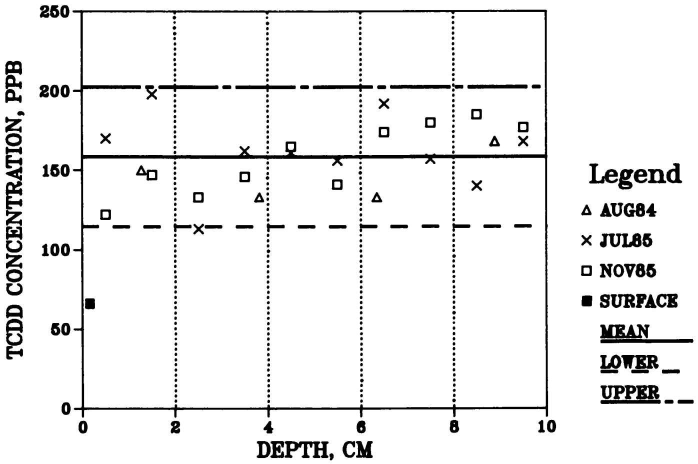


Figure 5. TCDD Concentration Profile for Control Side - Plot C

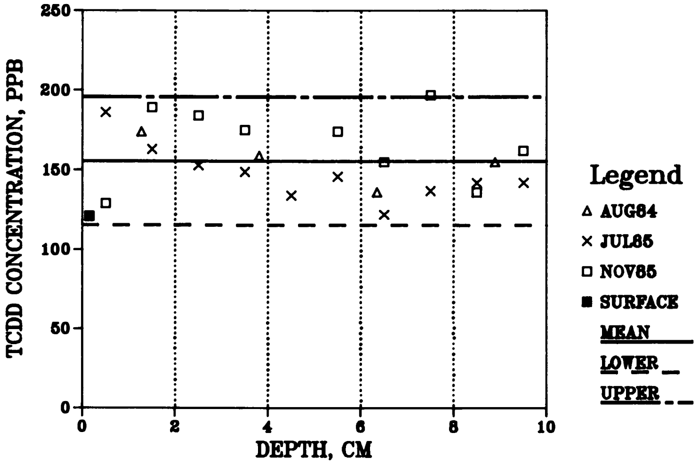


Figure 6. TCDD Concentration Profile for Shaded Side - Plot D

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch009

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch009

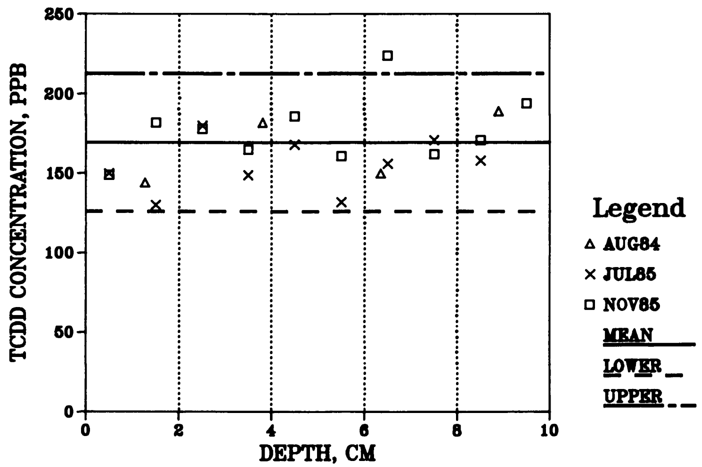


Figure 7. TCDD Concentration Profile for Unshaded Side - Plot D

lower than the average TCDD concentration of the soil layers below the 0 cm to 1 cm increment. The lower dashed line of Figures 3 thru 7 represents the lower 2 standard deviation limit from the mean of the plot TCDD concentration. Thus, the surface scraping TCDD concentrations are statistically lower than TCDD concentrations in the soil layers below. This result supports the TCDD vaporization and/or photodegradation hypothesis.

For the dry and shaded control experiment (Plot D - Shaded) the surface scraping sample concentration falls within the 2 standard deviation limits about the mean. This implies that no statistically significant loss of TCDD occurred in the dry shaded plot. Therefore, the presence of water and/or sunlight are important environmental parameters affecting the mobility of TCDD in soils.

The clean soil over dirty soil experiment (Plot C - Nursery) was designed to detect the movement of TCDD at very low rates. A 5 cm layer of TCDD free soil ($<$ 1 ppt) was applied to the surface of the plot. The layer of clean soil was separated from the dirty soil by a layer of new laboratory glass beads. TCDD was detected in all increments of the initially clean soil (Table I). Surface contamination by blowing dust could account for the surface concentrations of TCDD. Mixing of the dirty dirt below the glass beads with the clean layers above during sampling and sectioning would account for TCDD found above the glass bead layer. However, the simultaneous mixing of the glass beads would also be expected in the clean layers. This upward mixing of the clean glass beads did not occur. Downward sifting of the glass beads into the lower loose soil layers did occur. This downward mixing could have occurred during the initial application of the glass beads to the plots or during sampling. Some downward mixing of clean soil with dirty soil may be assumed to have occurred resulting in dilution of the lower soil increments TCDD concentrations. From this experiment, it may be concluded that TCDD is mobile.

Previous work by Freeman and Schroy (4) indicated that over 67 percent of the 2,3,7,8-TCDD contained in the top 1 cm of soil should volatilize during the first summer after application. The TCDD concentration in the top 1 cm of soil taken from experimental plots A and C-Control was found to have decreased by 20 percent and 18 percent respectively over the 15 months of elapsed time. The apparent rate of loss of TCDD from plots A and C-Control is lower than previously estimated by Freeman and Schroy (4). The TCDD concentration of the top 1 cm of soil taken from plot D-Shaded was found to have decreased 7 percent over the same 15 months. A loss of 7 percent is not statistically significant and it was concluded that plot D-Shaded remained unchanged over the course of the experiment.

Plant Study Results

All plant samples taken were analyzed for TCDD using the procedure presented above. Both the hexane surface rinses and the digested plant matter were found to contain TCDD (Table II). The surface rinses represent the TCDD 1) contained in dust adhering to the plants, 2) bound to the organic surface of the plants, and 3) contained in the outer cell layers of the plant. Many of the surface rinses had a green color indicating the presence of chlorophyll and the breaking of plant surface cells. Analysis of the plants taken from the Nursery side gave TCDD levels comparable to those found on

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch009

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch009

Table I. TCDD Concentration as a Function of Depth For Plot C - Nursery

Soil Increment cm	to cm	TCDD ppb	Frac. Rock	Frac. Glass	Sample Date Day Mon Yr
0.00	2.54	138.0	0.189	0.000	8 Aug 84 (1)
2.54	5.08	161.0	0.172	0.000	8 Aug 84 (1)
5.08	7.62	170.0	0.148	0.000	8 Aug 84 (1)
7.62	10.16	138.0	0.238	0.000	8 Aug 84 (1)
0.00	1.00	1.5	0.001	0.000	9 Oct 84
1.00	2.00	1.5	0.001	0.000	9 Oct 84
2.00	3.00	2.0	0.001	0.000	9 Oct 84
3.00	4.00	3.1	0.006	0.000	9 Oct 84
4.00	5.00	10.9	0.006	0.112	9 Oct 84
5.00	6.00	99.0	0.069	0.209	9 Oct 84
6.00	7.00	153.0	0.097	0.047	9 Oct 84
7.00	8.00	185.0	0.247	0.005	9 Oct 84
8.00	9.00	176.0	0.222	0.002	9 Oct 84
9.00	10.00	200.0	0.145	0.000	9 Oct 84
0.00	1.00	1.9	0.000	0.000	9 Jul 85
1.00	2.00	1.4	0.000	0.000	9 Jul 85
2.00	3.00	3.0	0.000	0.000	9 Jul 85
3.00	4.00	2.1	0.001	0.005	9 Jul 85
4.00	5.00	30.0	0.041	0.364	9 Jul 85
5.00	6.00	113.0	0.043	0.118	9 Jul 85
6.00	7.00	163.0	0.126	0.048	9 Jul 85
7.00	8.00	169.0	0.142	0.015	9 Jul 85
8.00	9.00	151.0	0.137	0.002	9 Jul 85
9.00	10.00	211.0	0.283	0.002	9 Jul 85
0.00	1.00	3.9	0.000	0.000	4 Nov 85
0.00	1.00	3.8 (2)	0.000	0.000	4 Nov 85
1.00	2.00	2.4	0.000	0.000	4 Nov 85
1.00	2.00	2.3 (2)	0.000	0.000	4 Nov 85
2.00	3.00	1.2	0.000	0.000	4 Nov 85
2.00	3.00	0.7 (2)	0.000	0.000	4 Nov 85
3.00	4.00	1.3	0.000	0.000	4 Nov 85
3.00	4.00	1.2 (2)	0.000	0.000	4 Nov 85
4.00	5.00	48.0	0.063	0.310	4 Nov 85
4.00	5.00	47.0 (2)	0.063	0.310	4 Nov 85
5.00	6.00	125.0	0.242	0.089	4 Nov 85
5.00	6.00	130.0 (2)	0.242	0.089	4 Nov 85
6.00	7.00	163.0	0.125	0.021	4 Nov 85
7.00	8.00	177.0	0.137	0.000	4 Nov 85
8.00	9.00	202.0	0.138	0.000	4 Nov 85
9.00	10.00	182.0	0.200	0.000	4 Nov 85

Notes: 1. These samples were taken before the 5 cm of nursery dirt was applied to the top of the plot on August 24, 1984. Thus, 5 cm should be added to the depths to correspond to the samples taken later.

2. Replicate Analysis

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch009

Table II. TCDD Content of Plant Matter Collected From Plots A, B, and C

Plot	Plant Wt. g	Plant TCDD ppb	Rinse TCDD (1) ppb	Total Plant ppb	Sample Date Day Mon Yr
A	739.0	0.378	0.139	0.517	9 Jul 85
A	-- (2)	0.940	0.138	1.078	9 Jul 85
A	7.05	1.228	0.749	1.977	4 Nov 85
B	1677.8	0.507	0.263	0.770	9 Jul 85
B	-- (3)	0.106	0.033	0.139	9 Jul 85
B	6.87	2.019	1.860	3.879	4 Nov 85
C - Control	1529.26	0.345	0.391	0.736	9 Jul 85
C - Control	6.4	1.111	0.851	1.962	4 Nov 85
C - Nursery	1188.6	0.158	0.029	0.187	9 Jul 85
C - Nursery	-- (4)	0.091	0.013	0.104	9 Jul 85
C - Nursery	2.61	1.243	0.839	2.082	4 Nov 85

Notes:

1. Rinse TCDD content reported as g TCDD x 10 9 per g plant matter (ppb by wt TCDD).
2. A total of 739 g of plant matter was collected from Plot A on July 9, 1985. This is a replicate workup and analysis for the TCDD concentration.
3. A total of 1677.8 g of plant matter was collected from Plot B on July 9, 1985. This is a replicate workup and analysis for the TCDD concentration.
4. A total of 1186.6 g of plant matter was collected from Plot C - Nursery on July 9, 1985. This is a replicate workup and analysis for the TCDD concentration.

Control side of Plot C. The TCDD concentrations in the nursery dirt (1 to 4 ppb) were approximately 30 times lower than the concentrations in the control side of Plot C. Thus, the TCDD was apparently drawn up through the plant roots and stored in the plant cells.

A TCDD material balance was done to determine the impact of the plant uptake of TCDD on the loss from the soil. It was assumed that all of the TCDD found in the plants came from a single 1 cm soil increment of density 1.5 g/cm3. Table III presents the results of this material balance calculation. The TCDD accumulation by the plants would change the soil concentration by less than 0.1 ppb over the time period of the field study. This is small change is well within the analytical and sampling error of this study and can be ignored as a significant TCDD loss mechanism. However, the possible movement of TCDD through the plants and the subsequent evaporation of the TCDD from the plant leaves can not be ruled out as a TCDD loss mechanism by this study.

Assuming no TCDD loss mechanisms in the plants (biodegradation, photodegradation, or vaporization from the leaves), allows the computation of a concentration factor (C.F.). The concentration factor was defined as the average plant concentration (total of plant and rinse data of Table II) divided by the average soil concentration of TCDD. The results of these calculations are given in Table IV. The concentration factor was found to vary widely with a minimum of 0.004 and a maximum of 0.038. These results are consistent with those of Facchetti (10) who found concentration factors of 0.0008 to 4 for maize and beans in field and greenhouse environments. Thus, in an area contaminated with TCDD (> 100 ppb) the concentration in plants and grasses may be assumed to be less than 4 percent of the soil concentration.

Conclusions

1. TCDD is mobile in soils in the field environment represented by typical Times Beach, Missouri soil.
2. Vaporization and/or photodegradation at the surface of a contaminated soil is a loss mechanism for TCDD.
3. The presence of water and/or sunlight is an important environmental parameter for the mobility of TCDD. Water appears to weaken the binding of TCDD to the soil matrix.
4. TCDD accumulation by the plants has a minimal impact on the soil concentration of TCDD. However, the possible movement of TCDD through the plants and the subsequent evaporation of the TCDD from the plant leaves can not be ruled out as a TCDD loss mechanism by this study.
5. Since this experiment was not explicitly designed to study the uptake of TCDD in plants, the mechanism for movement of TCDD through plants should be determined by a properly designed plant uptake experiment.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch009

Table III. Effect of Plant Matter TCDD Takeup on Soil TCDD Concentrations

Plot	Change in Soil TCDD Concentration ppb	Sample Date Day Mon Yr
A	0.0088	9 Jul 85
B	0.0114	9 Jul 85
C - Control	0.0336	9 Jul 85
C - Nursery	0.0052	9 Jul 85
A	0.0002	4 Nov 85
B	0.0004	4 Nov 85
C - Control	0.0004	4 Nov 85
C - Nursery	0.0002	4 Nov 85

Table IV. Bioconcentration of TCDD in Plant Matter

Plot	Plant TCDD Concentration ppb (1)	Soil TCDD Concentration ppb (2)	Concentration Factor (3)	Sample Date Day Mon Yr
A	0.80	100.15	0.008	9 Jul 85
B	0.45	101.82	0.004	9 Jul 85
C - Control	0.74	157.13	0.005	9 Jul 85
C - Nursery	0.19	-- (4)	---	9 Jul 85
A	1.98	100.15	0.020	4 Nov 85
B	3.88	101.82	0.038	4 Nov 85
C - Control	1.96	157.13	0.012	4 Nov 85
C - Nursery	1.09	-- (4)	---	4 Nov 85

[1]Average measured total plant matter concentration of TCDD from Table II.

[2]Average of all soil concentrations measured for plot in question.

[3]Concentration factor is defined as
C.F. = plant matter concentration/soil concentration

[4]Plot C - Nursery was initially covered with approximately a 5-cm clean nursery soil layer. The TCDD concentration below the nursery soil layer was found to be much higher than in the nursery soil layer. An average soil concentration will probably not describe the results and therefore was not reported.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch009

Acknowledgment

The authors wish to thank the Missouri Department of Natural Resources for their assistance at the Times Beach Dioxin Research Station. The authors also thank Sam Gibson, Robert Orth, Sally Walker, Jay Wendling, and Gary Thomas for their dedicated work in the analysis of the samples taken during this field study.

Literature Cited

1. Schroy, J.M.; Hileman, F.D.; Cheng, S.C. Aquatic Toxicology and Hazard Assessment: Eighth Symposium, ASTM STP 891, R.C. Bahner and D.J. Hansen, Eds., American Society for Testing and Materials, Philadelphia, 1985, p. 409-421.
2. Palausky, J.; Harwood, T.E.; Clevenger, S.; Kapila, S.; Yanders, A.F., Chlorinated Dioxins and Dibenzofurans in Perspective, C. Rappe, G. Choudhary, and L.H. Keith Eds., Lewis Publishers, Chelsea, Michigan, 1986, p. 211-224.
3. Jackson, D.R.; Roulier, M.H.; Grotta, H.M.; Rust, S.W.; Warner, J.S. Chlorinated Dioxins and Dibenzofurans in Perspective, C. Rappe, G. Choudhary, and L.H. Keith Eds., Lewis Publishers, Chelsea, Michigan, 1986, p. 185-200.
4. Freeman, R.A.; Schroy, J.M. Aquatic Toxicology and Hazard Assessment: Eighth Symposium, ASTM STP 891, R.C. Bahner and D.J. Hansen, Eds., American Society for Testing and Materials, Philadelphia, 1985, p. 422-439.
5. Young, A.L.; Thalken, C.E.; Arnold, E.L.; Cupello, J.E.; Cockerham, L.G. "Fate of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in the Environment", USAFA-TR-76-18, U.S. Air Force Academy, Colorado, NTIS, Rep, AD-A033491, 1976.
6. Liberti, A.; Brocco, D.; Allegrini, I.; Bertoni, G. in Dioxin: Toxicological and Chemical Aspects, Cattabeni, F., Cavallaro, A. and Galli, G., Eds. Medical and Scientific Books, New York, New York, 1978, p. 195-200.
7. Mill, T., "Prediction of the Environmental Fate of Tetrachlorodibenzodioxin," Paper presented at the Conference on Dioxins in the Environment, Michigan State University, December 6-7, 1983.
8. Yanders, A.F.; Kapila, S.; Schreiber, R.J. Chlorinated Dioxins and Dibenzofurans in Perspective, C. Rappe, G. Choudhary, and L.H. Keith Eds., Lewis Publishers, Chelsea, Michigan, 1986, p. 237-239.
9. Palausky, J.; Harwood, J.J.; Clevenger, T.E.; Kapila, S.; Yanders, A.F. Chlorinated Dioxins and Dibenzofurans in Perspective, C. Rappe, G. Choudhary, and L.H. Keith Eds., Lewis Publishers, Chelsea, Michigan, 1986, p. 211-223.
10. Facchetti, S.; Balasso, A.; Fichiner, C.; Frare, G.; Leoni, A.; Mauri, C.; Vasconi, M. Chlorinated Dioxins and Dibenzofurans in Perspective, C. Rappe, G. Choudhary, and L.H. Keith Eds., Lewis Publishers, Chelsea, Michigan, 1986, p. 225-235.

RECEIVED December 29, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch009

Chapter 10

Differential Bioavailability of 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin from Contaminated Soils

Thomas H. Umbreit, Elizabeth J. Hesse, and Michael A. Gallo

Department of Environmental and Community Medicine, University of Medicine and Dentistry of New Jersey, Robert Wood Johnson Medical School, Piscataway, NJ 08854

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch010

The bioavailability of TCDD from soils contaminated in the environment is an important determinant for risk assessment at TCDD contaminated sites. Literature reports indicate significant variation in bioavailability of TCDD from different sites. Soils from two of the major TCDD contaminated sites are compared for toxicity in guinea pigs and induction of aryl hydrocarbon hydroxylase (AHH) in rats. Times Beach, Missouri, soil is toxic and TCDD is highly bioavailable from this soil whereas Newark, New Jersey, soil is relatively non-toxic and has low bioavailability of TCDD. However, AHH induction in rats was approximately identical. These results confirm previous studies on bioavailability from these soils, and suggest that AHH induction may be an unreliable indicator of bioavailability.

The highly toxic compound 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) is one of a series of related chlorinated dioxins and furans. TCDD and 2,3,7,8-tetrachlorodibenzofuran (TCDF) are the most highly toxic members of the series. Brominated analogues appear to have similar toxicities to the chlorinated dioxins and furans. Structure activity studies have indicated that the 2,3,7,8 arrangement of halogens is required for maximum toxicity and receptor binding (1). TCDD is formed as a contaminant in the manufacture of the pesticide 2,4,5-T (2,4,5,-trichlorophenoxyacetic acid), which was a component of the herbicide Agent Orange in Vietnam. TCDD has entered the environment via use of contaminated herbicides (Vietnam, various locations in the United States), manufacturing procedures and

0097-6156/87/0338-0131$06.00/0
© 1987 American Chemical Society

accidents (Newark, New Jersey; Seveso, Italy; and others), and improper waste disposal (Times Beach and other locations in Missouri).

The major rationale for the cleanup of sites contaminated with TCDD is that the contamination by this highly toxic compound presents a major risk to human health. Risk is made up of the inherent hazard of the compound and the extent of exposure to the compound. Operationally, it has been assumed that most of the TCDD that can be detected by chemical analysis is available to cause toxicity. There has been little evidence until recently regarding the validity of this assumption. Several studies (discussed below) have suggested that the entire amount of TCDD that can be detected in a soil sample may not be completely bioavailable. Rather, some portion of the TCDD may be too tightly sorbed to the matrix to be released in biological systems. Further, the presence of other chemicals in the matrix may interfere with gastric uptake of TCDD or with the receptor mediated toxicity of TCDD.

Examination of the literature regarding bioavailability of TCDD from environmental materials (Table I) reveals several important points. (1). Bioavailability of TCDD appears to be quite variable, depending on the site and material tested, and the species used as an assay system. (2). Laboratory contaminated samples yield different reactions than environmentally contaminated samples. It appears that TCDD added to soil in the laboratory is much more bioavailable, at least initially, than TCDD on materials contaminated in the environment. (3). Apparently the amount of time TCDD has been in contact with the substrate greatly affects its bioavailability and chemical extractablility. This effect may be a physiochemical ageing or a weathering phenomena.

Poiger and Schlatter (2) reported a series of experiments on the absorption by animals of TCDD from various materials contaminated in the laboratory. The percentage of the TCDD dose (tritiated TCDD in ethanol) found in the liver of treated animals varied with the amount of time between dosing and assay, and the size of the dose of TCDD. They found that TCDD added to soil in the laboratory was about one quarter as bioavailable as the pure compound, and that this bioavailability was reduced (from 24% to 16%) by increasing the time of contact between soil and TCDD before dosing (from 24 hours to 8 days). They also reported that TCDD in a mixture with activated charcoal was only slightly bioavailable.

Isensee and Jones (3) detected little uptake by oats and soybeans of TCDD from sprayed soil, and found that adult plants took up even less than young shoots. Further, no uptake from leaves was detected. Van der

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch010

Table I: Comparison of Literature Reports of the Bioavailability of TCDD

author	type of sample	species	assay	bioavailability (uptake, toxicity)
Poiger + Schlatter 1980	soil or charcoal lab cont	rat	CA	charcoal <0.07% soil 10 hr = 24% soil 8 days = 16%
Isensee + Jones 1971	soil cont in lab	plant	CA	1.7%
van der Berg et al 1985	fly ash	g.p. hamster rat	CA	0.9-3.7% 1.7-2.4% <14%
Kuehl et al 1985a	fly ash	fish aquar	CA	0.024-2.1%
1985b	sediment	envir	CA	>37.5%
Silkworth et al 1982	PCB fire soot	g.p.	LD_{50}	80%
McConnell et al 1984	Times Beach soil	g.p. rat	CA AHH	60-85% 54-100%
Lucier et al 1986	Times Beach soil	rat	AHH	25-50%
Umbreit et al 1986a	Newark soil A soil B	g.p.	CA	 0.5% 21.3%
Cooper et al 1986	Newark soil	fish embryo	Tox	ca. 0.1%

Notes: g.p. = guinea pig. envir = environmental uptake. cont = contaminated. lab = laboratory. CA = chemical analysis. aquar = aquarium exposure. Tox = toxicity. AHH = aryl hydrocarbon hydroxylase induction in liver microsomes.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch010

Berg et al (4) reported that uptake of TCDD and other dioxins and furans from dietary fly ash was less than 14% in rats, 3.7% in guinea pigs, and 2.4 % in hamsters. Kuehl et al (5) showed a minimum of 37.5% uptake of TCDD from contaminated lake sediments into carp. In an aquarium study, Kuehl et al (6) found a maximum bioavailability of 2.1% to fish. Silkworth et al (7) demonstrated toxicity of soot from a PCB fire; however, the extremely low levels of TCDD in these materials make it difficult to relate these results to bioavailability of dioxins.

McConnell et al (8) studied the bioavailability of TCDD from the dioxin contaminated soils at Times Beach, Missouri. They reported a bioavailability (based on toxicity data and aryl hydrocarbon hydroxylase induction) of approximately 60-85% from soil from Times Beach, Missouri. Lucier et al (9) examined the same Missouri soils as reported by McConnell et al (8). Using AHH induction in rat liver as a measure, they estimated that TCDD was about 25-50% bioavailable from these soils. These data demonstrated a clear dose response for TCDD toxicity and AHH induction from the contaminated soils.

We previously reported studies of TCDD contaminated soils from a 2,4,5-T manufacturing site in New Jersey (10). No lethality was seen in guinea pigs treated with soils using doses as high as 12 ug TCDD/kg body weight. The LD_{50} in male guinea pig is ca. 0.6 ug TCDD/kg. Bioavailability was determined by analysis of liver samples and was estimated to be <0.5% from contaminated soil from the 2,4,5-T manufacturing site. A nearby metal scrap yard where the stills from the manufacturing plant had been broken down for scrap metal also had TCDD contamination in its soil. Tests of this soil demonstrated a bioavailability of TCDD in guinea pigs of ca. 21%. Cooper et al (11), examining these same soils, reported that embryos of the Japanese Medaka (*Oryzias latipes*) were extremely sensitive to toxicity from TCDD contaminated soils; however, the actual bioavailability of TCDD was still extremely low. We completed a study in susceptible mice of the long term and reproductive effects of the TCDD contaminated soils from New Jersey (12-14). Whereas female mice treated with TCDD in oil or TCDD added to decontaminated soil just before use were unable to reproduce, mice treated with TCDD contaminated soils reproduced successfully. Some reproductive toxicity was observed in females treated with contaminated soil; however, this toxicity could not be attributed to TCDD. The many other compounds present on these soils, including potent teratogens and fetotoxins, may well have caused the limited toxicities seen.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch010

Because the various reports of bioavailability have involved different protocols and systems, we undertook a study of both the Times Beach and the Newark soils.

METHODS

Soils were the identical samples previously studied, from Newark, New Jersey, (10-15) and Times Beach, Missouri (8,9) although they had been stored longer. TCDD (2,3,7,8 isomer) levels in the soils have been reported as approximately 2300 ppb at Newark and 770 ppb at Times Beach. Methods for the acute toxicity study of these soils in guinea pigs have been reported previously (15). Briefly, guinea pigs (ca. 200 g initially, 10 per sex per group) were given single doses of 10% soil suspensions in 5% aqueous gum Acacia by gavage and observed for up to 60 days following the single dose. Treatment materials included Times Beach soil, Newark soil, soil from Newark from which hydrocarbons had been stripped (designated "decontaminated soil"), and decontaminated soil to which pure TCDD in acetone was added shortly before use (designated "recontaminated" soil). Doses ranged from 1 to 10 ug TCDD/kg. All volumes administered were less than 15 ml. Animals that died during the course of the experiment were autopsied as soon as possible after death.

Rats were dosed with soil suspensions, by gavage, either once or for four consecutive days. Total TCDD dosages administered to rats were either 10 ug TCDD/kg or 40 ug/ TCDD/kg . Rats were sacrificed 24 hours after the final dose, autopsied, and hepatic microsomal fractions were collected. Aryl hydrocarbon hydroxylase (AHH) levels were determined in the microsomes, using the fluorescent assay of the product of the metabolism of benzo(a)pyrene to 3-OH benzo(a)pyrene (16).

RESULTS

Cause and mean time of death of guinea pigs in each treatment group was noted (summarized in Table II). Cause of death was determined by autopsy. Characteristic signs of TCDD intoxication included wasting, thymic atrophy and atrophy of mesenteric lymph nodes. Other causes of death (including pneumonia, abdominal fibrosis, and undeterminable) were approximately evenly distributed among treatment groups. Overall, the Times Beach soil was toxic (LD_{50} <10ug/kg) whereas the Newark soil was less toxic (LD_{05} >10ug/kg), when approximately the same TCDD dosages were given in the soil. Somewhat smaller doses of TCDD on recontaminated soil were much more toxic. Further, deaths occurred most rapidly in the recontaminated soil

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch010

group, and almost as fast in the group treated with Times Beach soil. The Newark soil death (1 death in 20 animals treated) occurred at the end of the observation period, and the designation based on autopsy of this death as induced by TCDD is questionable.

Table III presents the results of aryl hydrocarbon hydroxylase (AHH) determinations in rat liver microsomes. AHH levels were roughly equivalent for males and females in each group except the negative control. Times Beach soil and Newark soil induced AHH activity in both sexes (Times Beach soil was ca. 2 to 4 times decontaminated soil, while Newark soil was ca. 2 to 5.5).

DISCUSSION

The results of toxicity testing and liver analysis support the conclusions on bioavailability of TCDD from these soils previously presented by our group (10,12-15) for the Newark soil, and by McConnell et al for Times Beach soil (8). However, AHH induction in rats treated with Newark or Times Beach soil was equivalent.

Several possible reasons for the difference in bioavailability of TCDD from these soils can be suggested. 1) Differences in the composition of the soils may cause TCDD to be sorbed more tightly to one soil than to another soil [results of physiochemical characterization of the soils (unpublished) and of extractions of TCDD from the soils support this possibility (10)]. Therefore, it is less able to leave the soil and be taken up by the animal. The differences include the amount of highly absorptive material such as carbon particles or clay, the size distribution and total surface area of such particles, the amount and types of humic acids present, and moisture content. 2) The presence of various other compounds in the soil may affect TCDD binding to soil (such as solvents or oils) or interfere with TCDD binding to its receptor (such as other dioxins or furans). The Newark soil has been extensively analyzed and found to contain over fifty different dioxins and furans, some in concentrations almost as high as 2,3,7,8-TCDD. A wide variety of other compounds were also found. Although no detailed analysis of the Times Beach soil has appeared, unpublished results show a much more limited range of dioxins and furans present. Reports by Webster et al (17) and Jackson et al (18) suggest that dissolved and extractable organic matter influence leaching and transport potential of TCDD. 3) Contamination occurred by different mechanisms at the two sites: at Newark, TCDD contamination was the result of manufacturing processes, and the mixture reaching the soil contained a large aqueous component which percolated through the

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch010

Table II. Summary of the Time and Cause of Death of Guinea Pigs Treated With TCDD Contaminated Soils.

Treatment	TCDD ug/kg	TCDD Deaths %	time (days)
Decont soil	0	0	-
Newark soil	10	7	57
Times Beach soil	10	57	14.3±2.3
Recont soil	6	95	9.8±3.2

Notes: Recont soil = recontaminated soil, which was clean soil to which TCDD was added. Decont soil = soil from which chlorinated hydrocarbons had been removed. Days are expressed as mean +/- standard deviation

Table III. Induction of Aryl Hydrocarbon Hydroxylase (AHH) by TCDD Contaminated Soils in Rats.

Treatment	AHH Activity (pmoles/min/ug protein) mean	± SD	AHH/ background
Female 1 day			
decontaminated soil	0.042	0.057	1.00
recontaminated soil	0.560	0.070	13.33
Times Beach soil	0.173	0.100	4.12
Newark soil	0.218	0.156	5.19
Female 4 day			
decontaminated soil	0.058	0.047	1.00
recontaminated soil	0.580	0.150	10.00
Times Beach soil	0.212	0.030	3.66
Newark soil	0.315	0.114	5.43
Male 1 day			
decontaminated soil	0.115	0.060	1.00
recontaminated soil	0.420	0.140	3.62
Times Beach soil	0.240	0.100	2.07
Newark soil	0.230	0.110	1.98
Male 4 day			
decontaminated soil	0.143	0.070	1.00
recontaminated soil	0.650	0.150	4.55
Times Beach soil	0.310	0.090	2.17
Newark soil	0.370	0.120	2.59

Notes: 1 day dose was 10 ug TCDD/kg body weight. 4 day dose was 10 ug TCDD/kg body weight on each of 4 consecutive days for a total of 40 ug/kg. AHH was determined as pmoles/min/ug protein of 3-OH-benzo(a)pyrene formed, and reported as mean ± standard deviation of individual assays on four animals per treatment group. AHH/background = induction compared to decontaminated soil for each treatment group.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch010

soil for more than 10 years, while at Times Beach the TCDD contamination occurred because of the spreading of contaminated waste oil for dust control. It is possible that the TCDD at Times Beach is really dissolved in an oil matrix surrounding the soil particles, rather than bound to the soil. Such solution in oil would make the TCDD readily extractable and presumably more bioavailable.

Both Times Beach and Newark soils induced AHH activity in rats to similar levels despite the much lower toxicity of the Newark soil in guinea pigs. The amount of TCDD needed to cause AHH induction is considerably less than that needed to cause toxicity, and the low bioavailability of TCDD from the Newark soil may have been sufficient to induce AHH without being sufficient to cause toxicity. Another possible explanation of this result is the fact that among the numerous other compounds present on this soil were several potent AHH inducers [such as benzo(a)pyrene and benzo(a)anthracene (12)], that may have been bioavailable to a greater extent than TCDD. Importantly, the AHH induction in both groups implies that the use of AHH induction as a primary marker for TCDD exposure from environmental materials may be unreliable.

The results presented, and the previous reports confirmed by these results, demonstrate that soils from different TCDD contaminated sites have different toxicities and may represent quite different risks to human populations. The exact factors that influence the bioavailability of toxicants from soils remain to be elucidated.

Funded in part by USEPA cooperative agreement #CR812114-01-1.

Literature Cited

1. Safe, S.; Fujita, T.; Homonko, K.; Romkes, M.; Piskorska-Pliszczynska, J.; Denomme, M.A. Toxicologist 1986, 6, 309.
2. Poiger, H.; Schlatter, C. Food Cosmet. Toxicol. 1980, 18, 477-81.
3. Isensee, A.R.; Jones, G.J. J. Agr Food Chem. 1970, 19, 1210-14.
4. Van dan Berg, M.; de Vroom, E.; van Greevenbroek, M.; Olie, K.; Hutzinger, O. Chemosphere 1985, 14, 865-69.
5. Kuehl, D.W.; Cook, P.M.; Bateman, A.R. Fifth Internat. Symp. Chlorinated Dioxins, 1985, abstr. 172.
6. Kuehl, D.W.; Cook, P.M.; Bateman, A.R. Chemosphere 1985, 14, 871-72.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch010

7. Silkworth, J.; McMartin, D.; DeCaprio, A.; Rej, R.; O'Keefe, P.; Kaminsky, L.; Toxicol. Appl. Pharmacol. 1982, 65, 425-39.
8. McConnell, E.E.; Lucier, G.W.; Rumbaugh, R.C.; Albro, P.W.; Harvan, D.J.; Hass, J.R.; Harris, M.W. Science 1982, 223, 1077-79.
9. Lucier, G.W.; Rumbaugh, R.C.; McCoy, Z.; Hass, J.R.; Harvan, D.; Albro, P. Fund. Appl. Toxicol. 1986, 6, 364-71.
10. Umbreit, T.H.; Hesse, E.J.; Gallo, M.A. Science 1986, 232, 497-99.
11. Cooper, K.R.; Schell, J.; Umbreit, T.H.; Gallo, M.A.; submitted, Environmental Health and Toxicol.
12. Umbreit, T.H.; Hesse, E.J.; Gallo, M.A. submitted, Fund. Appl. Toxicol.
13. Umbreit, T.H.; Hesse, E.J.; Gallo, M.A. submitted, Fund. Appl. Toxicol.
14. Umbreit, T.H.; Hesse, E.J.; Gallo, M.A. in prep.
15. Umbreit, T.H.; Hesse, E.J.; Gallo, M.A. Chemosphere, 1986, in press.
16. Guengerich, F.P. In "Principles and Methods of Toxicology"; Hayes, A.W., Ed.; Raven Press: New York, 1982; pp. 615-16.
17. Webster, G.R.B.; Muldrew, D.H.; Graham, N.J.; Sarna, L.P.; Muir, D.C.G. 5th International Symposium on Chlorinated Dioxins and Related Compounds 1985, Bayreuth, FRG. abstract 84.
18. Jackson, D.R.; Roulier, M.R.; Grotta, H.M.; Rust, S.W.; Warner, J.S.; Arthur, M.F.; DeRoos, F.L. Land Disposal of Hazardous Waste. Barkley, N.P. Hazardous Waste Engineering Research Laboratory, Office of Res. and Dev., USEPA: Cincinnati, Ohio, 1985; pp. 153-168.

RECEIVED November 25, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch010

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

Chapter 11

Epidemiology of Populations Exposed to Dioxins

M. A. Fingerhut, M. H. Sweeney, W. E. Halperin, and T. M. Schnorr

National Institute for Occupational Safety and Health, Cincinnati, OH 45226

A historical review of studies of human exposure to dioxin-contaminated substances is presented, with emphasis on 2,3,7,8-tetrachlorodibenzodioxin (2,3,7,8-TCDD). Exposed populations include production workers, herbicide sprayers, persons exposed to Agent Orange, and residents of Missouri, U.S.A. and Seveso, Italy. Published medical reports have described chloracne, asthenia, neurological problems, and hepatotoxicity, but some of these studies have not had adequate comparison populations. Some recent epidemiologic studies have concluded that no outcomes could be attributed to dioxin exposure, but others have found associations of exposure with soft tissue sarcoma, lymphoma, nasal and stomach cancer. These studies are described.

This paper presents a historical review of reports and clinical studies of humans exposed to dioxin-contaminated substances. Dioxins are chemical contaminants produced during the synthesis of chlorophenols and certain other halogenated aromatic hydrocarbons. There are 75 dioxin isomers of which 2,3,7,8 tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) is the most toxic in animal studies (1). Table I illustrates some major substances contaminated with isomers of dioxin. These manufactured products include 2,4,5-trichlorophenol (TCP) and its derivatives, such as 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), which are contaminated with 2,3,7,8-TCDD, as well as pentachlorophenol, which is contaminated with hexa-, hepta-, and octachlorinated isomers of dioxin.

This chapter not subject to U.S. copyright.
Published 1987, American Chemical Society

TABLE I. The Relevant Substances

MANUFACTURED PRODUCT	DIOXIN CONTAMINANT
2,4,5-TRICHLOROPHENOL 2,4,5-T(ACID, ESTER, AMINE) SILVEX (ACID, ESTER, AMINE)	2,3,7,8-TETRACHLORODIBENZODIOXIN (2,3,7,8-TCDD)
PENTACHLOROPHENOL	HEXACHLORODIBENZODIOXINS HEPTACHLORODIBENZODIOXINS OCTACHLORODIBENZODIOXINS
2,4-D	NONE DICHLORODIBENZODIOXINS TRICHLORODIBENZODIOXINS TETRACHLORODIBENZODIOXINS (1,3,6,8 or 1,3,6,9)

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

Figure I illustrates some major events which have occurred since production of the chlorophenols began in the United States in the mid-1930's . Although accidents have occurred periodically and adverse health effects related to dioxin exposure have been recognized in exposed workers, it was not until the 1970's that the problems received public attention. During that decade, animal studies demonstrated the severe toxicity of 2,3,7,8-TCDD and it was realized that large numbers of people had potential exposure to 2,3,7,8-TCDD through the use of Agent Orange in the Vietnam war, the distribution of dioxin-contaminated wastes throughout Missouri, U.S.A. and the contamination of several communities after a major explosion in Seveso, Italy. This perception of a potential human crisis motivated rapid advances in analytic techniques, animal toxicology and the initiation of epidemiologic investigations of human health effects.

The Early Period: Descriptive Medical Reports (1937-1978)

From 1935 to 1978, workers world-wide were exposed during periodic explosions at trichlorophenol production facilities as well as during their daily production activities. Table II illustrates some major industrial accidents which yielded published reports of health effects. Table III lists some major health outcomes reported following exposures to products contaminated with 2,3,7,8-TCDD. The primary dermal effect of exposure is chloracne, a persistent form of acne caused by exposure to a number of chlorinated organic compounds. Chloracne occurred in some exposed persons in all accidents, as well as in children in Seveso (2-4). Chloracne appeared with and without other health effects and persisted in some persons up to 28 years after exposure (5-6).

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

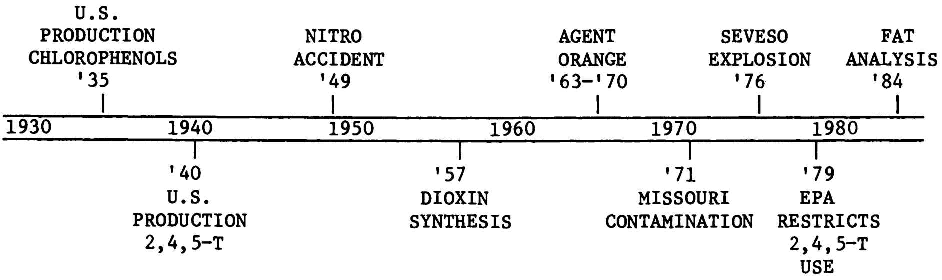


FIGURE 1. Key Events in the History of Dioxin Exposures

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

TABLE II. Industrial Explosions

YEAR	LOCATION
1949	MONSANTO, NITRO, W.V., U.S.A.
1953	BASF LUDWIGSHAFEN, W. GERMANY
1963	PHILIPS-DUPHAR AMSTERDAM, NETHERLANDS
1966	RHONE POULENC GRENOBLE, FRANCE
1968	COALITE & CHEMICALS PROD. BOLSOVER, DERBYSHIRE, U.K.
1976	ICMESA SEVESO, ITALY

Neurologic effects, including disturbances of both the peripheral and central nervous systems, were reported following every accident and in many industrial exposures. The neurologic effects almost always included profound weakness and fatigue, as well as pain and weakness in extremities. Other reported neurologic symptoms included headaches, irritability, sleep disturbances, peripheral neuropathy, depression, confusion, and personality changes (7-8).

Hepatotoxic effects were also reported in exposed workers. These included hepatitis, hepatomegaly, alterations in level of liver enzymes and porphyria cutanea tarda, a disturbance of liver metabolism in which excess porphyrins are produced and excreted. Some symptoms of porphyria cutanea tarda were exhibited as dermal effects, including hyperpigmentation, blistering, and an abnormal growth of hair on the face and body, also known as hirsutism (7,9-10).

Less frequently reported effects included gastrointestinal disturbances, renal, cardiovascular and respiratory problems, and abnormalities in lipid levels (11-13).

Case reports in 1977 and in 1978 from a clinic in Sweden noted that neoplasms (soft tissue sarcoma and lymphoma) were observed more frequently than expected in workers exposed to chlorophenols or phenoxy herbicides (14-15).

In a unique situation, three British laboratory workers who synthesized 2,3,7,8-TCDD in a laboratory experienced chloracne, asthenia, neurologic and gastrointestinal problems along with changes in lipid metabolism (12). Their medical effects were of special interest because they seem to have been associated only with pure 2,3,7,8-TCDD.

TABLE III. Major Human Health Effects Following 2,3,7,8-TCDD Exposure, 1937-1978

SYSTEM	EFFECT
DERMAL	CHLORACNE
NEUROLOGIC	PERIPHERAL NEUROPATHIES FATIGUE DEPRESSION PERSONALITY CHANGES
HEPATIC	HEPATITIS ENLARGED LIVER ABNORMAL ENZYME LEVELS PORPHYRIA CUTANEA TARDA

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

Adverse health outcomes were also described among community residents in Missouri and Seveso (2,16). Although the conditions of exposure of workers and the community residents differed, some of the reported health outcomes in these groups were quite similar (17).

In summary, many medical reports were published between 1937 and 1979 which described serious problems in multiple organ-systems among individuals exposed to 2,3,7,8-TCDD. However, during this 50 year period of exposure, no formal studies were conducted which included a control group of non-exposed persons.

It is generally assumed that the affected workers experienced heavy exposures. Extrapolation to less severely exposed populations from these reports was difficult because of the absence of comparison groups of non-exposed persons, and because limited information was provided about severity of the exposure and the proportions of persons injured.

The Current Period: Epidemiologic Studies (1979-1986)

The observations in the medical reports just described provided the hypotheses for controlled epidemiologic studies of exposed human populations. With the exception of one study of herbicide workers in 1974 (18), epidemiologic studies have been published only since 1979. In the United States and in some other countries, most industrial exposures to 2,3,7,8-TCDD had ceased by 1979 when restrictions on uses of 2,4,5-T were issued by the U.S.

Overview of Basic Epidemiologic Study Designs. Epidemiology is the systematic study of the distribution and determinants of disease in human populations (19). Analytic research studies fall into several designs such as cohort studies, case-control studies, proportional mortality ratio (PMR) studies and cross sectional studies. The choice of a particular study design is determined by the

characteristics of the problem under investigation. However, all designs include requirements for accurate assessment of exposure, adequate sample size to detect an effect, and appropriately selected control groups. Although studies of human populations often cannot have an experimental design as strict as that of toxicologic studies of animals, a goal in designing epidemiologic studies is quite similar: to construct as much as possible a study group and a control group which differ only with respect to the variable of interest. Often the researcher can account for many confounding variables, such as age and sex, in the design or in the analysis of the study.

In a cohort mortality study design, the study population is chosen on the basis of exposure to a particular substance. The mortality rates in the exposed group are compared to mortality rates in an unexposed group. Often the unexposed comparison group is a standard population, such as the U.S. male population or an appropriate unexposed group such as unexposed workers in the same plant.

In a case-control design, the study population is selected on the basis of disease status of the individual cases. Controls are selected who are free of the disease of interest at the time of the study. To determine whether the disease is related to the exposure of interest, the frequency of the exposure is compared among cases and controls. Usually the cases and controls (or their next of kin) are interviewed about occupational, and environmental exposures as well as personal habits. Of particular concern in a case-control study is the correct or unbiased ascertainment of relevant exposure. Unintentional or intentional under-reporting or over-reporting of exposure by either the cases or the controls may produce erroneous results.

In proportional mortality ratio (PMR) studies, the observed proportions of deaths due to particular causes are compared to expected proportions calculated from the comparison group. There are two major limitations of this type of study. First, one must assume that the deaths studied are representative of the larger group, which may not be true. Second, proportions must add to one so a deficit in one cause of death category will necessarily produce excesses in other categories.

Epidemiologic studies which evaluate the long term effect of exposure on health generally have had a cross-sectional study design, in which the medical assessment of current workers or residents is made at one point in time. In such a design, one major concern is that sick individuals may not be included because they left the workplace or area before the time of the cross-sectional medical study.

In all epidemiologic studies, the statistical power is of special concern. A study of adequate statistical power has the ability to detect an excess outcome as statistically significant, if the excess is truly present. Studies which have too few subjects may be unable to detect an excess simply because the size of the study group is too small. Therefore, in studies with few subjects, excesses over the expected should not be ignored solely because the result was not statistically significant.

American Chemical Society
Library
1155 16th St., N.W.
Washington, D.C. 20036

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

In evaluating the results of an epidemiologic study, it is important to examine the extent of exposure of the subjects. "Exposure" is defined by the researchers and is specific only to that study. For example, in some cohort studies of chemical workers the exposure has been defined as assignment to the TCP or 2,4,5-T production processes. In other studies, the definition of exposure has been less specific; for example, in some cohort studies of herbicide sprayers, all sprayers have been considered "exposed" even though the authors have no knowledge whether the various herbicides sprayed by a particular individual included 2,4,5-T.

Cohort Studies. To date, a number of cohort studies have been conducted of chemical workers from the U.S. and Europe and of herbicide sprayers from Sweden and Finland (20-28). In general, the studies have not demonstrated a definitive relationship between mortality from any malignant or nonmalignant condition and exposure to chlorophenols or phenoxy herbicides. All of the studies lacked sufficient statistical power due to the small number of individuals in each cohort.

Table IV presents the cohort studies of chemical workers. In two cohort studies of U.S. chemical workers, statistically nonsignificant excesses in mortality were observed for the ICD categories describing cancers of the lung, soft tissue sarcoma, stomach, male genital organs and for non-Hodgkins lymphoma (21,23). One soft tissue sarcoma death was observed in the Zack study (21), but the meaning of one death is difficult to interpret. The Cook study (23) updated two earlier studies, so those are not shown here (22,29).

Among European chemical workers, a statistically significant increase in deaths from stomach cancer was observed in German workers exposed to trichlorophenol (24). Among Danish women workers exposed to phenoxy herbicides, a statistically nonsignificant excess in the incidence of cervical cancer was found. Male workers exposed to phenoxy herbicides showed a statistically significant excess of lung cancer as well as an elevated but statistically nonsignificant excess of rectal cancer. A single case of soft tissue sarcoma was also observed (25). An excess incidence of STS which was not statistically significant, was found also among manual service workers, a group including maintenance and shipping workers who may have had exposure to phenoxy herbicides. One case of STS was observed also among workers assigned to departments without phenoxy herbicides. Interpretation of these results was severely hampered by the limited exposure assessment.

Cohort studies of herbicide sprayers are presented in Table V. An excess of mortality from stomach cancer was statistically significant in one small Swedish study (27). No specific malignancies were found in excess in the other studies. All had low statistical power and limited exposure assessments. The researchers had insufficient information about the types of phenoxy herbicides to which the workers were exposed and limited data about the intensity and duration of the exposures. Some phenoxy herbicides do not contain 2,3,7,8-TCDD. Corroborating results from other studies and additional observations obtained through further vital status follow-up of the cohorts may reduce the ambiguity of the results.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

TABLE IV. Cohort Studies of Chemical Workers

POPULATION	EXPOSURE	DEATHS/N	CANCER EXCESSES	OBS/EXP	CONCERN
Mortality Studies					
U.S.A. (21)	TCP	32/121	LUNG	5/2.85	P
			STS	1/?	
U.S.A. (23)	TCP 2,4,5-T	298/2189	STOMACH	5/3.2	P
			MALE GENITAL ORGANS	6/3.8	
			NON-HODGKINS LYMPHOMA	5/2.1	
W. GERMANY (24)	TCP	21/74	STOMACH	3/0.6**	P
Incidence Studies					
DENMARK (25)	PH Women	0/240	CERVIX	5/1.82	
	PH Men	0/690	LUNG	11/5.33**	P,E
			RECTAL	4/1.49	
			STS	1/0.50	
	MS Men	0/988	STS	3/0.6	
	O Men	0/1385	STS	1/0.8	

N, TOTAL NUMBER IN COHORT; P, POWER; E, EXPOSURE ASSESSMENT; OBS/EXP, OBSERVED/EXPECTED; TCP, TRICHLOROPHENOL; STS, SOFT TISSUE SARCOMA; PH, PHENOXY HERBICIDES; MS, MANUAL SERVICE WORKERS; O, OTHER MANUFACTURING WORKERS; **p lt 0.05

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

TABLE V. Cohort Mortality Studies of Herbicide Sprayers

POPULATION	EXPOSURE	DEATHS/N	CANCER EXCESSES OBS/EXP	CONCERN
SWEDEN (27)	PH	45/348	STOMACH 3/.71**	P,E
SWEDEN (26)	2,4,5-T	29/158	NO	P,L,E
FINLAND (28)	2,4-D 2,4,5-T	144/1926	NO	P,L,E

P, POWER; L, LATENCY; E, EXPOSURE ASSESSMENT; PH, PHENOXY HERBICIDES; N, TOTAL NUMBER IN COHORT; OBS/EXP, OBSERVED/EXPECTED; **p lt 0.05

Case-Control Studies. The case control studies presented in Table VI were conducted to assess the possible association between the occurrence of soft tissue sarcoma, lymphoma, nasal and colon cancers with exposure to phenoxy herbicides and chlorophenols. No statistically significant association was found between colon cancer and such exposures (30), but an association was found with lymphoma (31). Nasal cancer was found to be associated with exposure to phenoxy herbicides and chlorophenols in a Swedish study (32). However, a Danish study found no association between nasal cancer and exposure (33). Differences in results between the studies may be explained by the methods used for control selection. The Swedish controls were selected from the general population and the Danish controls were selected from among other cancer cases. Some critics have suggested that as a group, cancer cases have heightened recall of past exposures, thus increasing the likelihood of a positive finding unless the controls have a similar heightened recall.

In the past few years, there has been widespread interest in the possibility that individuals exposed to phenoxy herbicides and chlorophenols may be at greater risk of developing soft tissue sarcomas. Soft tissue sarcomas constitute a category of very rare cancers with a total mortality rate of 1 STS per 100,000 persons per year in the United States (34). The soft tissue sarcomas are malignant neoplasms of diverse histologic subtypes, which occur throughout the body in mesenchymal connective tissue other than bone (35). Because of the diversity of histologic subtypes, these malignancies are often not reported accurately on death certificates (36) and may not be recognized accurately by general pathologists (37). These factors may lead to the incorrect reporting of soft tissue sarcomas.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

TABLE VI. Case-Control Studies

STUDY	DISEASE	EXPOSURE	CASES/ CONTROLS	EXCESSES	CONCERN
SWEDEN (30)	COLON	PH, CL	157/541	NO	E
SWEDEN (31)	LYMPHOMA	PH CL	169/338	5-FOLD** 4-FOLD**	E
SWEDEN (32)	NASAL	PH CL	71/541	2-FOLD 7-FOLD**	E
DENMARK (33)	NASAL	CL	146/2465	NO	E
SWEDEN (38)	STS	PH, CL	52/206	5-FOLD**	E
SWEDEN (39)	STS	PH, CL	110/220	6-FOLD**	E
NEW ZEALAND (40)	STS	PH CL	82/92	NO 5-7 FOLD** (PELT HANDLERS)	E
New York, USA (41)	STS	VIETNAM SERVICE AO	281/410	NO	E

PH, PHENOXY HERBICIDES; CL, CHLOROPHENOLS; AO, AGENT ORANGE; E, EXPOSURE ASSESSMENT; P, POWER; **p lt 0.05

Several studies have assessed the plausibility of the association between soft tissue sarcomas and exposure to phenoxy herbicides and chlorophenols. Table VI lists two case-control studies conducted in Sweden, which selected controls from the general population and concluded that exposure to the herbicides or to chlorophenols resulted in a greater than 5-fold excess in soft tissue sarcomas (38-39). A New Zealand study found no association with exposure to phenoxy herbicides (40). This study drew controls from other cancer cases in the National Cancer Registry of New Zealand. However, in the New Zealand study, a 5-fold excess of STS was noted in persons who handled animal pelts, which are sometimes preserved with trichlorophenols containing isomers of dioxin.

Because of possible exposure to Agent Orange during tours of duty in southeast Asia, Vietnam Veterans have been concerned that they may have increased risk of developing soft tissue sarcomas. Agent Orange was composed of equal parts of esters of 2,4,5-T and 2,4-D. Table VI presents results from an interview study of soft tissue sarcoma cases from the New York State Cancer Registry with controls selected from New York driver's license registration files (41). Greenwald et al. found no association between STS and

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

a history of Vietnam service or reported exposures to Agent Orange. The major limitation of the study was the assessment of exposure of individuals to Agent Orange.

Studies Using National Census Data. Table VII lists four studies with different epidemiologic designs which have utilized existing computerized population data to evaluate soft tissue sarcoma. This record-based type of study is valuable for generating hypotheses, but generally provides weak evidence for confirming hypotheses because of the absence of individual exposure information. Three of the studies used broad occupational categories such as farmer or forester and assumed that exposure to phenoxy herbicides occurred to the group (42-44). Milham examined proportions of deaths due to STS which were recorded on death certificates in Washington State (42) and Balajaran used occupations recorded upon registration of the cancer in the National Cancer Register of England and Wales (43). Each found excess STS over expected in a category of farmer. Wiklund matched Swedish census data for occupation to cancers in the cancer registry. He found no excess soft tissue sarcoma in 6 categories of agricultural and forestry workers (44). Kogan studied causes of death listed on death certificates of veterans who died in Massachusetts between 1972 and 1973 and found that the proportion of deaths due to soft tissue sarcoma and to kidney cancer was significantly higher among veterans who served in Vietnam when compared to those who served elsewhere (45).

TABLE VII. Studies of Soft Tissue Sarcoma from National Census Data

POPULATION	EXPOSURE	STUDY SIZE	EXCESSES (STUDY)	CONCERN
WASHINGTON STATE DEATHS (42)	FARMER	429,926	STS 1.5 FOLD** (PMR)	E
ENGLAND AND WALES (43)	FARMER	1961/1961	STS 1.7 FOLD** (CC)	E
SWEDEN (44)	FARMER FORESTER	354,620	NO (COHORT)	E
MASS. DEATHS 1972-73 (45)	VIETNAM SERVICE	840	STS 5 FOLD** KIDNEY 4 FOLD** (PMR)	E

E, EXPOSURE; PMR, PROPORTIONAL MORTALITY RATIO STUDY; CC, CASE-CONTROL STUDY; **p lt 0.05.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

In summary, studies of STS have reported both positive and negative findings. All of the studies had limitations in statistical power or assessment of exposure. To date, no definitive answer exists as to whether STS is associated with exposure to phenoxy herbicides or chlorophenols. The situation is similar for lymphoma, stomach and nasal cancer.

Reproductive Outcome Studies. Studies of miscarriages and birth defects have been few in number and have considered male-mediated effects by examining pregnancy outcomes among the wives of exposed men (46-50). These studies are listed in Table VIII. The studies of Vietnam veterans had uncertain exposure data and considerable time between last exposure and conception. One study found no excess birth defects among Vietnam veterans (48). In two studies, excesses were noted of some birth defects: neural tube defects and cleft lip (47,50). These excesses appeared to be associated with probable Agent Orange exposure. However, the significance of the findings is questionable because of the large numbers of statistical tests performed. The reproductive studies among the wives of occupationally exposed men observed no excess of adverse reproductive outcomes. These studies had the potential for simultaneous occurrence of exposure and conception. However, the power of the studies is quite low and exposure assessments are inadequate (46,49).

TABLE VIII. Reproductive Studies

POPULATION	COMPARISON	EXPOSURE	EXCESSES (STUDY)	CONCERN
NEW ZEALAND SPRAYERS 548 (46)	AGRICULTURAL CONTRACTORS 441	2,4,5-T	NO (COHORT)	E,P
WIVES CHEMICAL WORKERS 370 (49)	WIVES CHEMICAL WORKERS 345	2,4,5-T TCP	NO (COHORT)	E
ANATOMICAL BIRTH DEFECTS 5,000 (47)	NORMAL BIRTHS ATLANTA 3,000	VIETNAM	SPINA BIFIDA** CLEFT LIP** (CC)	E
BIRTH DEFECTS AUSTRALIA 8517 (48)	NORMAL BIRTHS AUSTRALIA 8517	VIETNAM SERVICE	NO (CC)	E
RANCH HAND PERSONNEL 1024 (50)	USAF PERSONNEL 1024	AO	NEURAL TUBE DEFECTS+ (COHORT)	E,P

E, EXPOSURE ASSESSMENT; P, POWER; AO, AGENT ORANGE; ** p lt 0.05;
+ BASED ON PRELIMINARY ANALYSIS

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

Cross-Sectional Medical Studies. Table IX lists the cross-sectional studies. In a study of active, retired, and former workers exposed to TCP and 2,4,5-T, Singer observed a statistically significant correlation between reduced nerve conduction velocity and length of employment (51). Bond found excesses of ulcer and digestive system diseases in a survey of company bi-annual medical examinations (52). Four cross-sectional studies were conducted in chemical workers who had been exposed at least ten years prior to the study. Suskind and Moses separately studied U.S. workers at a West Virginia plant (53-54). Suskind (53) observed excesses of peptic ulcer, decreased libido, reduced lung function in current smokers, and 3 cases of Peyronie's disease, a condition of internal scarring of the penis. In the study by Moses (54), all subjects had been exposed to 2,4,5-T. The group which had experienced chloracne had increased levels of triglycerides and a liver enzyme, as well as decreased sensation to pinprick, decreased libido and increased sexual dysfunction.

Pazderova examined Czech workers about ten years after exposure and found sustained neural and psychiatric problems and arterio-sclerosis in the Czech workers, as well as glucose intolerance and increases in cholesterol (55). In a laboratory study of British workers ten years after exposure, Martin found increases in some liver enzymes as well as increased levels of cholesterol and triglycerides in exposed workers (56). Whether a health risk results from the enzyme and lipid changes is not known, but one concern is that lipid abnormalities may confer increased risk of coronary vascular disease.

Several medical studies are presented in Table X which have been conducted in groups outside of the industrial sector. In a medical study of exposed Seveso subjects, the prevalence of peripheral neuropathy was three times higher among those exposed persons with chloracne (57). In a study of Missouri residents from a mobile home park with soil contamination with 2,3,7,8-TCDD, some parameters of immune response were reported to differ between the exposed and unexposed (58). The U.S. Air Force completed a comprehensive medical study of the personnel of Operation Ranch Hand, the individuals who sprayed Agent Orange in Vietnam (50). Increased prevalence of enlarged livers and an increase of basal and squamous cell carcinomas was observed.

In summary, the epidemiologic medical studies conducted after 1979 have had limitations such as inadequate exposure assessment, limited participation, or limited control of confounding factors, such as age and socioeconomic status of the comparison group. Consequently, they have not provided definitive conclusions regarding the persistence of medical effects of exposure to phenoxy herbicides and chlorophenols or the risk of disease among populations with low-level exposures. They do suggest that chloracne, ulcer, neurologic disease and elevations of triglycerides, cholesterol and liver enzymes persist in some exposed persons.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

TABLE IX. Medical Studies of Chemical Workers

AUTHOR	EXPOSURE	EXCESSES	CONCERN
(51)	TCP 2,4,5-T	SLOWED NERVE CONDUCTION VELOCITIES	PN, CG
(52)	TCP 2,4,5-T	ULCER (2,4,5-T) DIGESTIVE SYSTEM DISEASES (2,4,5-T)	PN
(53)	TCP	PEPTIC ULCER PEYRONIE'S DISEASE REDUCED LUNG FUNCTION DECREASED LIBIDO	E,PN
(54)	2,4,5-T	TRIGLYCERIDES ALTERED LIVER ENZYMES DECREASED SENSATION DECREASED LIBIDO SEXUAL DYSFUNCTION	E,PN
(55)	TCP	POLYNEUROPATHY ARTERIOSCLEROSIS PSYCHIATRIC DISORDERS GLUCOSE INTOLERANCE CHOLESTEROL	CG
(56)	TCP	D-GLUCARIC ACID GAMMA GLUTAMYL TRANSFERASE DECREASED SERUM CALCIUM CHOLESTEROL, TRIGLYCERIDES	E

TCP, TRICHLOROPHENOL; E, EXPOSURE ASSESSMENT; PN, PARTICIPATION; CG, COMPARISON GROUP

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

Table XI lists several current epidemiologic studies which improve upon earlier design limitations such as statistical power and exposure assessment. Researchers at the National Institute for Occupational Safety and Health (NIOSH) are nearing completion of a large cohort mortality study of 7,000 U.S. chemical workers from 14 companies which produced trichlorophenol, 2,4,5-T and pentachlorophenol. A particular asset of this study is the availability of good information on type and duration of exposure of the workers. The International Agency for Research on Cancer (IARC) is preparing a worldwide registry of data on chemical workers and sprayers with exposure to phenoxy herbicides and chlorophenols. The NIOSH data base will be included in the IARC International Dioxin Registry. Case control studies are also underway for soft tissue sarcoma and lymphoma at the National Cancer Institute (NCI), Veterans Administration (VA) and the Centers for Disease Control (CDC). These studies will assess possible associations of these cancers with use of phenoxy herbicides in Kansas, with Vietnam service, and with exposure to Agent Orange.

Table XII lists medical studies to be completed in the next few years. NIOSH researchers will conduct a comprehensive medical examination study of chemical workers who made trichlorophenol and 2,4,5-T, and Northwestern University is conducting a medical study of workers who made pentachlorophenol. Good exposure information is available for both of these studies.

TABLE X. Non-Industrial Medical Studies

AUTHOR	POPULATION/ COMPARISON	EXPOSURE	EXCESSES	CONCERN
(57)	+/- CHLORACNE 319/380	TCP	PERIPHERAL NEUROPATHY	E
(58)	MISSOURI 154/155	TCDD	ANERGY T CELL RATIO	E,P
(50)	RANCH HAND/ USAF PERSONNEL 1024/1024	AO	ENLARGED LIVER NON-MELANOTIC SKIN CANCER	E

TCP, TRICHLOROPHENOL; TCDD, 2,3,7,8-TCDD; AO, AGENT ORANGE; E, EXPOSURE ASSESSMENT; P, POWER

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

TABLE XI. Current Epidemiological Studies

STUDY	AGENCY*	POPULATION	COMPARISON	EXPOSURE
COHORT MORTALITY	NIOSH	CHEMICAL WORKERS 7,000	U.S. MALE POPULATION	PHENOXY HERBICIDES CHLOROPHENOLS
DIOXIN REGISTRY	IARC	CHEMICAL WORKERS/SPRAYERS 17,000	NATIONAL POPULATIONS	PHENOXY HERBICIDES CHLOROPHENOLS
CASE/ CONTROL	NCI	STS 200 LYMPHOMA 400	KANSAS POPULATION	PHENOXY HERBICIDES
CASE/ CONTROL	VA	STS 250	PATHOLOGY 750	VIETNAM AO
CASE/ CONTROL	CDC	STS 400 LYMPHOMA 1,300	NATIONAL POPULATION 1,300	VIETNAM AO

* Full names are found in text.

TABLE XII. Current Medical Studies

AGENCY	POPULATION	COMPARISON	EXPOSURE	COMPLETION
NIOSH	CHEMICAL WORKERS 450	NEIGHBORS 450	2,4,5-T TCP	1989
NORTH-WESTERN UNIV.	CHEMICAL WORKERS 500	WORKERS 500	PCP 2,4,5-T	1987
USAF	RANCH HAND PERSONNEL 1024	AIR FORCE PERSONNEL 1024	AGENT ORANGE	PHASE II 1987
CDC	AO EXPOSED VIETNAM VETS 2000	VIETNAM VETS NOT EXPOSED 2000	AGENT ORANGE	1989
CDC	VIETNAM VETS 2000	NON-VIETNAM VETS 2000	VIETNAM SERVICE	1989

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

To assess the potential health effects from exposure to Agent Orange, the Air Force is conducting the second phase of the Ranch Hand Health Study, and a large medical study of army veterans is underway at the Centers for Disease Control. The comparison groups for these studies are non-exposed military personnel.

Conclusions

What conclusions can be drawn from the epidemiologic studies of persons exposed to dioxin-contaminated products?

1. Epidemiologic studies have been conducted within a very short time, between 1979 and 1985. All have been limited in exposure assessment or statistical power.
2. Chloracne is a consequence of exposure to dioxin in some people and is persistent for many years in some workers.
3. Acute medical consequences following exposure to dioxin-contaminated substances in industrial accidents included peripheral neuropathy, weakness, cardiovascular disease, liver dysfunction, abnormalities in liver enzymes and lipid levels. Symptoms of these medical consequences have persisted in some workers when examined in several studies conducted many years after exposure. No clear understanding yet exists of the severity or persistence of these health problems. Additionally, there is no clear information regarding medical risks for populations with low exposures to phenoxy herbicides and chlorophenols.
4. To date, exposure to phenoxy herbicides and chlorophenols has been shown in some studies to be associated with soft tissue sarcoma, lymphoma, stomach cancer, and nasal cancer, but no outcome has been conclusively established or rejected.
5. Some current studies have better statistical power and improved designs for assessing exposure. Recent successes with chemical analyses of human adipose tissue suggest that it may soon be possible to analyze biological levels of 2,3,7,8-TCDD in human adipose tissue, or better yet, in serum. Success in these efforts could contribute greatly to the accuracy of current studies of health outcomes in exposed persons.

LITERATURE CITED

1. Esposito MP, Tierman TO, and Dryden FE: Dioxins, EPA-600/2-80-197, November, 1980.
2. Reggiani, G. Medical Problems Raised by the TCDD Contamination in Seveso, Italy., Arch Toxicol 40:161-188 (1978).
3. Butler MG: Acneform dermatosis produced by Ortho (2 chloro-phenyl) Phenol Sodium and Tetrachlorophenol Sodium. Arch Derm Syph 35: 251-254, 1937.
4. Kimmig J and Schulz KH: Occupational chloracne caused by aromatic cyclic ethers. Dermatologica 115: 540-546, 1957.
5. Goldman PJ: Critically acute chloracne caused by trichloro-phenol decomposition products. Arbeitsmed Sozialmed Arbeit-shygiene 7:12-18, 1972.
6. May G,: Chloracne from the accidental production of tetrachlor-odibenzo-dioxin. Br J Ind Med 30:276-283, 1973.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

7. Bleiberg J, et al: Industrially acquired porphyria. Arch Dermatol 89:793-97, 1964.
8. Jirasek L, et al: Chronic poisoning by 2,3,7,8-tetrachlorodibenzo-p-dioxin. Cesk Dermatol 49:145-157, 1974.
9. Cholak J, Schafer LJ, Yeager D: "Reports on clinical and environmental surveys at Monsanto Chemical Co. Nitro, West Virginia, 1953." Department of Environmental Health, University of Cincinnati, Unpublished.
10. Strik JJTWA: The occurrence of chronic-hepatic porphyria in man caused by halogenated hydrocarbons. In: "Chemical Porphyria in man"; Strik JJTWA and Koeman JH (eds); Elsevier/North-Holland: New York, 1979.
11. Bauer H, et al: Industrial poisoning in the manufacture of chlorophenol compounds. Arch Gewerbepath Gewerbehyg 18:538-555, 1961.
12. Oliver RM: Toxic effects of 2,3,7,8-tetrachlorodibenzo 1,4 dioxin in laboratory workers. Br J Ind Med 32:49-53, 1975.
13. Poland A, et al: A health survey of workers in a 2,4-D and 2,4,5-T plant. Arch Environ Health 22:316-27, 1971.
14. Hardell L: Malignant mesenchymal tumors and exposure to phenoxy acids-a clinical observation. Lakartidningen 74:2853, 1977.
15. Hardell L: Soft tissue sarcomas and exposure to phenoxyacetic acids-a clinical observation. Lakartidningen 74:2753-2754, 1977.
16. Kimbrough RD, Carter CD, Liddle JA and Cline, RE. Epidemiology and pathology of a tetrachlorodibenzodioxin poisoning episode. Arch Environ Health 32:77-85, 1977.
17. IARC. "Long Term Hazards of Polychlorinated Dibenzodioxins and Polychlorinated Dibenzofurnas". IARC Technical Report No 78/001. International Agency for Research on Cancer. Lyon, 1978.
18. Axelson O and Sundell L: Herbicide exposure, mortality and tumor incidence: an epidemiological investigation on Swedish railroad workers. Work Environ Health 11:21-28, 1974.
19. Mausner JS and Kramer S: "Epidemiology - An Introductory Text". W.B. Saunders Co., Philadelphia, 1985.
20. Zack J and Suskind R: The mortality experience of workers exposed to tetrachlorodibenzodioxin in a trichlorophenol process accident. J Occup Med 22:11-14, 1980.
21. Zack JA and Gaffey WR: A mortality study of workers employed at the Monsanto Company plant in Nitro, West Virginia. Environ Sci Res 26:575-591, 1983.
22. Cook R, Townsend J, Ott MG and Silverstein L: Mortality experience of employees exposed to 2,3,7,8-tetrachlorodibenzodioxin (TCDD). J Occup Med 22:530-532, 1980.
23. Cook RR, Bond GG, Olson RA, Ott MG, and Gondek MR: Evaluation of the mortality experience of workers exposed to the chlorinated dioxins. Fifth International Conference on Dioxin, Bayreuth, West Germany, 1985.
24. Thiess AM, Frentzel-Beyme R, and Link R: Mortality study of persons exposed to dioxin in a trichlorophenol-process accident that occurred in the BASF AG on November 17, 1953. Am J Ind Med 3, 179-189, 1982.
25. Lynge E: A follow-up study of cancer incidence among workers in manufacture of phenoxy herbicides in Denmark. Br J Cancer 52:259-270, 1985.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

26. Hogstedt C. and Westerland B: Cohort study of causes of death of forestry workers with and without exposure to phenoxy acid preparations. Lakartidningen 77:1828-1831, 1980.
27. Axelson O, Sundell L, Andersson K, Edling C, Hogstedt C, and Kling H: Herbicide exposure and tumor mortality. Scand J Work Environ Health 6:73-79, 1980.
28. Riihimaki V, Asp S, and Hernberg S: Mortality of 2,4-dichlorophenoxyacetic acid and 2,4,5-trichlorophenoxyacetic acid herbicide applicators in Finland. Scand J Work Environ Health 8:37-42, 1982.
29. Ott MG, Holder BB, Olson RD: A mortality analysis of employees engaged in the manufacturer of 2,4,5-trichlorophenoxyacetic acid. J Occup Med 22:47-50, 1980.
30. Hardell L: Relation of soft tissue sarcoma malignant lymphoma and colon cancer to phenoxy acids or chlorophenol and other agents. Scand J Work Environ Health 7:119-130, 1981.
31. Hardell L, Eriksson M, Lenner P, and Lundgren E: Malignant lymphoma and exposure to chemicals, especially organic solvents, chlorophenols and phenoxy acids: a case control study. Br J Cancer 43:169-176, 1981.
32. Hardell L, Johansson B, Axelson O: Epidemiological study of nasal and nasopharyngeal cancer and their relation to phenoxy acid or chlorophenol exposure. Am J Ind Med 3:247-257, 1982.
33. Olsen JH and Jensen OM: Nasal cancer and chlorophenols. Lancet 2:47-48, 1984.
34. Young JL, Percy CL, and Asire AJ, et al: SEER: Incidence and Mortality Data, 1973-1977. Nat Cancer Inst Monograph 57:1-1082, 1981.
35. Russell WO, Cohen J, Enzinger F, Hajdu S, Heise H, Martin RG, Meissner W, Miller WT, Schmitz RL, Suit HD: A clinical and pathological staging system for soft tissue sarcomas. Cancer 40:1562-1570, 1977.
36. Percy C, Stanek E, and Gloekler L: Accuracy of cancer death certificates and its effect on cancer mortality statistics. Am J Pub Health 71:242-250, 1981.
37. Fingerhut MA, Halperin WE, Honchar PA, Smith AB, Groth DH, and Russell WO: An evaluation of reports of dioxin exposure and soft tissue sarcoma pathology in U.S. chemical workers. Scand J Work Environ Health 10:299-303, 1984.
38. Hardell L and Sandstrom A: Case-control study: soft tissue sarcoma and exposure to phenoxyacetic acids or chlorophenols. Br J Cancer 39:711-717, 1979.
39. Eriksson M, Hardell L, Berg N, Moller T, and Axelson O: Soft tissue sarcomas and exposure to chemical substances: a case-referent study. Br J Ind Med 38:27-33, 1981.
40. Smith AH, Pearce NE, Fisher DO, Giles HJ, Teague CA, and Howard JK: Soft tissue sarcoma and exposure to phenoxyherbicides and chlorophenols in New Zealand. JNCI 73:1111-1117, 1984.
41. Greenwald P, Kovasznay B, Collins DN, Therriault G: Sarcomas of soft tissues after Vietnam service. JNCI 75:1107-1109, 1984.
42. Milham S: Herbicides, occupation, and cancer. Lancet i:1464-1465, 1982.
43. Balarajan R and Acheson E: Soft tissue sarcomas in agriculture and forestry workers. J Epidemiol Comm Health 38:113-116, 1984.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

44. Wiklund K and Holm L: Soft tissue sarcoma risk in Swedish agricultural and forestry workers. JNCI 76:229-234, 1986.
45. Kogan M and Clapp R: Mortality among Vietnam veterans in Massachusetts 1972-1983. Abstract, Am J Epidemiol 122:523, 1985.
46. Smith A H, et al: Congenital defects and miscarriages among New Zealand 2,4,5-T sprayers. Arch Environ Health 37:197-200, 1982.
47. Erickson JD, Mulinare J, McClain PW, et al: Vietnam veterans' risks for fathering babies with birth defects. JAMA 252: 903-912, 1984.
48. Donovan JW, Adena MA, Rose G, Battistutta D: "Case-Control Study of Congenital Anomalies and Vietnam Service (Birth Defects Study)". Report to the Minister for Verterans' Affairs. Australian Government Publishing Service, Canberra, 1983.
49. Townsend JC, Bodner KM, VanPeenen PF, Olson RD, and Cook RR: Survey of reproductive events of wives of employees to chlorinated dioxins. Am J Epidemiol 115:695-713, 1982.
50. Lathrop GD, Wolfe WH, Albanese RA, Moynahan PM: "An epidemiologic investigation of health effects in air force personnel following exposure to herbicides: Baseline morbidity study results." U.S. Air Force School of Aerospace Medicine, Aerospace Medical Division, Brooks Air Force Base, Texas, 1984.
51. Singer R, et al: Nerve conduction velocity studies of workers employed in the manufacture of phenoxy herbicides. Environ Res 29:297-311, 1982.
52. Bond GG, Ott MG, Brenner FE, and Cook RR: Medical and morbidity surveillance findings among employees potentially exposed to TCDD. Br J Ind Med 40:318-324, 1983.
53. Suskind RR and Hertzberg VS: Human health effects of 2,4,5-T and its toxic contaminants. JAMA 251:2372-2380, 1984.
54. Moses M, Lilis R, Crow KD, Thornton J, Fischbein A, Anderson HA, and Selikoff IJ: Health status of workers with past exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin in the manufacture of 2,4,5-trichlorophenoxyacetic acid: comparison of findings with and without chloracne. Am J Ind Med 5:161-182, 1984.
55. Pazderova-Vejlupkova J, et al: The development and prognosis of chronic intoxication by tetrachlorodibenzo-p-doxin in men. Arch Environ Health 36:5-11, 1981.
56. Martin JV: Lipid abnormalities in workers exposed to dioxin. Br J Ind Med 41:254-256, 1984.
57. Filippini G, Bordo B, Crenna P, Massetto N, Musicco M, and Boeri R: Relationship between clinical and electrophysiological findings and indicators of heavy exposure to 2,3,7,8-tetrachlordibenzo-dioxin. Scand J WorK Environ Health 7:257-262, 1981.
58. Hoffman RE, Stehr-Green PA, Webb KB, et al: Health Effects of long-term exposure to 2,3,7,8-tetrachlorodibenzodioxin. JAMA 255:2031-2038, 1986.

RECEIVED November 25, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch011

Chapter 12

Human Breast Milk Levels of Dioxins and Dibenzofurans: Significance with Respect to Current Risk Assessments

Arnold Schecter[1] and Thomas A. Gasiewicz[2]

[1]Department of Preventive Medicine, Clinical Campus at Binghamton, State University of New York, Health Science Center at Syracuse, 88 Aldrich Street, Binghamton, NY 13903

[2]Environmental Health Sciences Center, Department of Biophysics, University of Rochester Medical Center, Rochester, NY 14642

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch012

While the partitioning of the lipophilic chlorinated dibenzodioxins and dibenzofurans into breast milk fat represents a mode of excretion and detoxification for the lactating female, this may not be without impact on nursing infants. This study examined whether the levels of these compounds in human breast milk from Vietnam and North America exceeded the recommended intake values currently employed by the United States Environmental Protection Agency and the United States Public Health Services Centers for Disease Control. Based on a number of assumptions, these calculations suggest that many nursing infants from the south of Vietnam may have consumed, and are continuing to consume, amounts of these contaminants that are much greater than currently recommended intake values. Although concentrations of these compounds in mother's milk samples from North America are much lower, they are still near or slightly greater than the recommended amounts. Analyses of milk samples from areas of northern Vietnam showed levels of contamination that are below employed standards. Further study is needed to determine if there is any real short- or long-term clinical risk to the nursing infants in these populations.

Elevated human tissue dioxin levels found in certain areas after environmental contamination were described in previous publications (1-3). The highest levels were noted in human breast milk lipid a few years following heavy 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) contamination from herbicides, especially Agent Orange, sprayed in Vietnam. Elevated levels in this tissue from individuals living in the south of Vietnam decreased with time but were still above levels observed in samples from the North American continent (3). It was hypothesized that the relatively low human tissue levels of dioxins observed in individuals living in the north of Vietnam may be attributed

0097-6156/87/0338-0162$06.00/0
© 1987 American Chemical Society

to the absence of use of synthetic chemicals in that region and to the fact that no spraying of dioxin contaminated herbicide occurred in the north. Previous results also presented evidence of penta- through octa-chlorinated dioxin contamination of human tissue in the south of Vietnam and North America that was suggestive of the characteristic isomer pattern seen in technical grade pentachloro-phenol (PCP), a commonly used wood preservative (3).

In the present study we estimated whether the intake of dioxin and dibenzofuran contaminated breast milk presents a potential risk to the health of the nursing infants in these populations. This was performed using "virtually safe" or "acceptable daily intake" values which various governmental agencies in the United States currently employ in their risk assessments for these compounds.

Methods and Assumptions

The recommended daily intake values as estimated by the United States Environmental Protection Agency (EPA) (4) and the Centers for Disease Control (CDC) (5,6) have been used for the present calculations. For the EPA the "Acceptable Daily Intake" (ADI) for 2,3,7,8-TCDD is 1×10^{-12} g/kg/day. This value is based on the lowest-observed-adverse-effect-level (LOAEL) of 1×10^{-9} g/kg/day for a reproductive effect in rats (7,8), a 10-fold uncertainty factor because a LOAEL is used as the basis of the calculation rather than a no-observed adverse effect level (NOAEL), and an additional uncertainty factor of 100 based on the existence of lifetime animal studies and lack of knowledge of the effects in man. (4). For a 10^{-6} (1/1,000,000) cancer risk, the EPA has estimated a 95% lower-limit criteria for a lifetime intake of 6.3×10^{-15} g/kg/day (9). For this same cancer risk, the CDC has estimated a "virtually safe dose" of 28×10^{-15} g/kg/day (hereafter, CDC1) (5,6). These estimates are based on a linear-derived multistage extrapolation model that is usually used for genotoxic agents such as ionizing radiation. However, the results to date indicate that 2,3,7,8-TCDD is a tumor promoter rather than an initiator (10,11). Therefore, the CDC estimate for a 10^{-6} cancer risk of 636×10^{-15} g/kg/day (hereafter CDC2), which is based on a non-linear model for tumor promoters (5,6), has also been consided. The present paper takes no position as to the validity of these values, but cites them as figures which appropriate government agencies have published or prepared for regulatory purposes or to make health risk estimates.

For the calculation of the relative intake of chlorinated dioxins (PCDDs) and dibenzofurans (PCDFs) by a nursing infant, some of the assumptions that have been made include: 1) the infant is breast-fed for 1 year, 2) the average infant weight for 1 year is 10 kg (12), 3) 100% of the dioxin and dibenzofuran isomers are absorbed through the gastrointestinal tract of the infant, and 4) approximately 3% of human breast milk is fat (2,3,13). For the % of lipid in adipose tissue, we have used the mean of determined values for the individual populations under study. For the population in North America we have assigned a value of 80% (2), while for the south and north Vietnam populations these values are 62% and 56%, respectively (3). We have also used a value of 850 ml/day for the average amount of milk consumed (12). A number of studies have reported values from 650 to 1000 ml/day for infants 1 to 8 months of age (12-15).

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch012

Values previously reported (1-3,16,17) for concentrations of dibenzodioxin and dibenzofuran isomers in breast milk either measured directly or calculated from adipose tissue were used for these analyses. For the sake of clarity some of these values are reported here again.

Results

Recalling levels of the PCDD and PCDF isomers previously reported in adipose tissue from a North American population (Tables I-II) (16,17), the most toxic isomer, 2,3,7,8-TCDD, is found in the lowest concentration, whereas the octachlorodioxin is found in the highest concentration. We and others have reported similar results of analysis of separate sample populations in the United States (1,18,19). These results emphasize the need to consider other dioxin and dibenzofuran isomers, in addition to the 2,3,7,8-TCDD isomer, when an evaluation of potential human health risk is performed.

Table II indicates the qualitative and quantitative similarity of the dioxin and dibenzofuran isomers in human milk on a lipid basis as compared to those present in adipose tissue. The 4.5 ppt value for 2,3,7,8-TCDD in milk derived from a pooled sample from 200 mothers is close to the mean value of 7.1 ppt of 2,3,7,8-TCDD from individual adipose tissue samples. The lower mean value in breast milk compared to adipose tissue may be due to the lack of inclusion of the non-detectable values from individual adipose tissue specimens in calculating the mean. For adipose tissues the geometric mean of the positives for 2,3,7,8-TCDD is 6.0 ppt and the arithmetic mean is 4.4 if calculated by including the nondetected specimens at a value of one-half the detection level. The other isomer values are comparatively similar for fat or milk, except for the octachlorodibenzo-p-dioxin which, for unknown reasons, is markedly lower in the milk than in the adipose samples from these two unmatched populations. When reported on a lipid basis, the similarity of these values in milk and adipose tissue is expected because of the lipophilic nature of these compounds. This relationship has been reported for other lipid soluble chemicals as well (20). Similar levels of these isomers in human breast milk samples from European populations have been reported recently (21-3).

Table III summarizes some of the reported and calculated levels of PCDD and PCDF isomers in human breast milk from populations in the south and north of Vietnam (3), and the United States (16). It is noteworthy that analysis of 9 adipose tissue samples taken in 1984 from individuals living in the north of Vietnam showed no detectable concentrations of 2,3,7,8-TCDD at a detection limit of 2 ppt (3). As expected, the milk pattern and levels on a lipid basis are similar to that reported for adipose tissue (2,3,16) (Tables I and II).

Many health organizations world-wide are currently estimating risks of the halogenated dibenzo-p-dioxins and dibenzo-furans to humans in terms of "2,3,7,8-TCDD equivalents," i.e., the amount of the specific chemical or mixture which would cause the same degree of toxicity at 2,3,7,8-TCDD. Tables IV and V present values currently assigned to various PCDD and PCDF isomers found in human tissues in an attempt to relate them to 2,3,7,8-TCDD (24-6). These values are subject to change, and may well be assigned different numerical values as new toxicology studies provide more data and toxic end points upon which to provide a less speculative basis of assigment. The concept of toxic equivalents

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch012

TABLE I
Average Concentration of PCDD and PCDF Isomers in Adult Human Adipose Tissue from a North American Population[a]

Isomer	Concentration[b]	Range[b]
2,3,7,8-TCDD	5.0	(ND-10)
1,2,3,7,8-PeCDD	32	(ND-180)
HxCDD	72	(7.9-330)
1,2,3,4,7,8,9-HpCDD	87	(ND-390)
OCDD	560	(64-1250)
2,3,7,8-TCDF	9.1	(ND-32)
2,3,4,7,8-PeCDF	27	(ND-77)
HxCDF	18	(2.9-35)
1,2,3,4,6,7,8-HpCDF	18	(ND-55)
OCDF	60	(ND-360)

[a] Data from (16)
[b] Values as the mean of 46 composite samples from 900 individuals, as pg/g, or parts per trillion (ppt).

TABLE II
PCDDs and PCDFs in Human Milk and Adipose Tissue from a Canadian Population

	Milk[a]		Adipose Tissue[a]
Isomer	Wet Wt. Basis	Lipid Basis	Lipid Basis
2,3,7,8-TCDD	0.17	4.5	7.1[b]
1,2,3,7,8-PeCDD	0.38	10	11
HxCDD	2.6	69	97
1,2,3,4,6,7,8-HpCDD	5.3	138	151
OCDD	10.3	271	951
2,3,7,8-TCDF	0.16	4.2	ND (2)
2,3,4,7,8-PeCDF	0.44	12	17
HxCDF	0.46	12	14
1,2,3,4,6,7,8-HpCDF	0.50	13	36
OCDF	ND (0.2)		ND (4)

[a] Results for milk are from a single pooled sample of 200 human milk samples collected across Canada in 1981. Results for human adipose tissues from Canada in 1976 from 46 accident victims. Data from (17). Data are presented as mean values for adipose tissue samples, pg/g.

[b] Average of 25 positive out of 46 samples; geometric mean is 6.0 and the arithmetic mean is 4.4 pg/g if NDs are estimated at one-half the detection limit.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch012

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch012

TABLE III
Reported and Calculated Concentration of PCDDs and PCDFs in Human Milk on a Wet Weight or Whole Milk Basis

Population	Isomer	Concentration[a] Mean[b]	Concentration[a] Ranges	Reference for Original Data
south of Vietnam (1973)	2,3,7,8-TCDD	4.2	(2.3-6.9)	(3)
	2,3,4,7,8-PeCDF	0.7	(ND-0.8)	
	OCDD	7.9	(3.4-12)	
south of Vietnam (1984)	2,3,7,8-TCDD	0.68	(ND-3.1)	(3)
	1,2,3,7,8-PeCDD	0.44	(ND-1.3)	
	HxCDD	3.0	(0.7-10.4)	
	1,2,3,4,6,7,8-HpCDD	5.3	(0.41-21.3)	
	OCDD	39.8	(4.2-102.3)	
	2,3,4,7,8-PeCDF	0.63	(0.13-1.4)	
	HxCDF	1.7	(0.41-5.0)	
	1,2,3,4,6,7,8-HpCDF	0.9	(0.13-2.2)	
north of Vietnam (1984)	2,3,7,8-TCDD	ND		(3)
	1,2,3,7,8-PeCDD	.039	(ND-0.11)	
	HxCDD	0.24	(ND-0.72)	
	1,2,3,4,6,7,8-HpCDD	0.59	(ND-1.7)	
	OCDD	2.8	(ND-6.2)	
	2,3,4,7,8-PeCDF	0.31	(ND-0.88)	
	HxCDF	0.32	(ND-0.6)	
	1,2,3,4,6,7,8-HpCDF	0.09	(ND-0.32)	
United States	2,3,7,8-TCDD	0.19	(ND-0.38)	(16)
	1,2,3,7,8-PeCDD	1.2	(ND-6.8)	
	HxCDD	2.7	(0.3-12.4)	
	1,2,3,4,7,8,9-HpCDD	3.3	(ND-14.6)	
	OCDD	21	(2.4-46.9)	
	2,3,7,8-TCDF	0.34	(ND-1.2)	
	2,3,4,7,8-PeCDF	1.0	(ND-2.9)	
	HxCDF	0.68	(0.11-1.31)	
	1,2,3,4,6,7,8-HpCDF	0.68	(ND-2.1)	
	OCDF	2.3	(ND-13.5)	

[a] Values are as mean, pg/g, (range). Results from the south of Vietnam (1973) are reported values (3). Other values presented have been calculated from previously published data and using reported values for the % of lipid in adipose tissue as 80, 62, and 56 for the United States (1,2), south of Vietnam (3), and north of Vietnam (3), respectively, An estimated value of 3% lipid in human milk was also used.

[b] None detectibles were included in the calculation of arithmetic means as one-half of the detection limits.

TABLE IV
Estimation of Relative Toxicities (2,3,7,8-TCDD Equivalents) of Various Chlorinated Dibenzo-p-dioxins

Isomer	EPA (24)	New York State (25)	California (26)
2,3,7,8-TCDD	1	1	1
Other TCDDs	0.01	0	0
2,3,7,8-PeCDDs	0.5	0.5	1
Other PeCDDs	0.005	0	0
2,3,7,8-HxCDDs	0.04	0.03	1
Other HxCDDs	0.0004	0	0
2,3,7,8-HpCDDs	0.001	0	1
Other HpCDDs	0.00001	0	0
OCDD	0	0	1

TABLE V
Estimation of Relative Toxicities (2,3,7,8-TCDD Equivalents) of the Chlorinated Dibenzofurans

Isomer	EPA (24)	New York State (25)	California (26)
2,3,7,8-TCDF	0.1	0.33	1
Other TCDFs	0.001	0	0
2,3,7,8-PeCDFs	0.1	0.33	1
Other PeCFs	0.001	0	0
2,3,7,8-HxCDFs	0.01	0.01	1
Other HxCDFs	0.0001	0	0
2,3,7,8-HpCDFs	0.001	0	1
Other HpCDFs	0.00001	0	0
OCDF	0	0	0

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch012

assumes that all of the isomers have a similar mechanism of action and that total "dioxin" toxicity may be calculated by adding the weighted factors for the individual isomers present. Table VI shows the result of simple arithmetic calculations going from measured PCDD and PCDF values to their "dioxin equivalents." Although the value of 2,3,7,8-TCDD remains 5.0, the value of total equivalents varies from 27.8 to 35.3 to 828, depending on the weighting values assigned to the various isomers. The ratio of added risk for all of the PCDD and PCDF isomers compared to 2,3,7,8-TCDD alone varies from 5.6. to 7.1 to 166, again based on the reported levels in adipose tissue from individuals in the United States. This uncertainty or lack of agreement between governmental agencies must be considered when any conclusions as to the health consequences of these compounds in breast milk are presented. Notably, the penta- and not the tetra-chlorinated dioxins and dibenzofurans appear to give the greater amount of toxic equivalents compared to all other isomers.

Table VII compares the calculated average total dioxin toxic equivalents in human breast milk (or extrapolated from adipose tissue data) from populations in Vietnam and the United States. These numbers are based on the EPA equivalency values shown in Tables IV and V. For purposes of comparison and since the equivalent value for the north of Vietnam is the lowest we know of world-wide at the present time, we have defined this level as one. On this basis, the levels in the United States are on the average approximately 16-fold higher, while the values for south of Vietnam for 1973 and 1984 are approximately 67- and 17-fold higher respectively. It should be noted that the values for the south of Vietnam would be higher still were the data from the breast milk samples collected in 1970 (27) included in these calculations. By either calculation, there existed a population in the south of Vietnam and nursing during the early 1970's, whose consumption of these contaminants in breast milk was quite high, and higher than any other population of nursing infants. Values from industrial countries such as the United States, Canada (Table II), or in Europe (21-23) are well above those seen in such nonindustrialized areas as the north of Vietnam, but below what was seen in heavily contaminated areas such as the south of Vietnam in 1973.

Using the average values for the total dioxin equivalents (Table VII) the daily intake of these equivalents by a nursing infant can be estimated (Table VIII). Notably, all values are considerably greater than the ADI of one pg/kg/day from the USEPA, and even much greater than the recommended daily intakes for 10^{-6} risk of cancer as estimated by the EPA or the CDC. However, these recommended intakes have been estimated considering a lifetime of exposure to the contaminants. If it is assumed that the infants in the United States nurse for 1 year, then during this year the child will have consumed above the lifetime (70 years) recommended intake, the factor of excess being from 2 to 201 fold, depending on the standard and the agency cited. If it is further assumed that the child has no further intake (or excretion) of these contaminants from other sources for the remainder of his/her lifetime, and the weight increases to 70 kg (i.e., a dilution of the body burden of dioxin equivalents), then these factors of excess will be from 0.3 to 29. Table VIII also presents results of similar calculations for the Vietnam populations, using however a 55 kg body weight attained at 70 years of age. Thus, for a 1973 nursing infant

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch012

TABLE VI
Estimation of Total "2,3,7,8-TCDD Equivalents" in Adipose Tissue from Residents of the United States

Isomer	Mean Concentration (pg/g)[a]	2,3,7,8-TCDD Equivalents EPA	NYS	Cal
2,3,7,8-TCDD	5.0	5.0	5.0	5.0
1,2,3,7,8-PeCDD	32	16	16	32
HxCDD	72	2.9[b]	2.2[b]	72[b]
1,2,3,4,7,8,9-HpCDD	87	0.87	0	87
OCDD	560	0	0	560
2,3,7,8-TCDF	9.1	0.91	3.0	9.1
2,3,4,7,8-PeCDF	27	2.7	8.9	27
HxCDF	18	0.18[b]	0.18[b]	18[b]
1,2,3,4,6,7,8-HpCDF	18	.018	0	18
OCDF	60	0	0	0
Total 2,3,7,8-TCDD Equivalents		27.8	35.3	828
Ratio of Total Equivalents/2,3,7,8-TCDD Alone		5.6	7.1	166

[a] Data from (16), Table I.
[b] Assuming all HxCDDs and HxCDFs are as the 2,3,7,8-isomer. Similar values for 2,3,7,8-HxCDD and 2,3,7,8-HxCDF have been reported (1-3).

TABLE VII
Comparison of Total 2,3,7,8-TCDD Equivalents in Human Milk in Vietnam and United States

Population	Total 2,3,7,8,-TCDD Equivalents (pg/g)[a]
south of Vietnam (1973)	4.27
south of Vietnam (1984)	1.11
north of Vietnam (1984)	0.064
United States	1.04

[a] Calculated from data presented in Table III and using the USEPA equivalency values presented in Tables IV and V.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch012

TABLE VIII
Estimated Average Daily and First Year Intake of Total 2,3,7,8-TCDD Equivalents by Nursing Infants from Vietnam and the United States, and the Ratio of these First Year Intakes to the Recommended Lifetime Intake Values for 2,3,7,8-TCDD

Population	Daily Intake[a] (pg/kg/ day)	First Year Intake (pg/kg)	Ratio of First Year Intake to Recommended Lifetime Intake Values		
				No Age[c] Correction	Age[d] Correction
south of Vietnam (1973)	362	132,477	EPA[b]	828	150
			CDC1[b]	184	33
			CDC2[b]	8	1.5
south of Vietnam (1984)	94.4	34,456	EPA	215	40
			CDC1	54	9.1
			CDC2	2.2	0.4
north of Vietnam (1984)	5.4	1,971	EPA	12	2.2
			CDC1	3.1	0.5
			CDC2	0.1	.03
United States	88.4	32,266	EPA	201	29
			CDC1	50	6
			CDC2	2	0.3

[a] Calculated from data in Table VII.

[b] For a cancer risk of 10^{-6} the lifetime (70 year) recommended intake values of 2,3,7,8-TCDD are 1.6×10^{-10}, 6.4×10^{-10} and 1.6×10^{-8} g/kg for EPA, CDC1 and CDC2, respectively. See Methods and Assumptions.

[c] Assuming 10 kg infant.

[d] Assuming growth to a 70 kg adult for United States and a 55 kg adult for Vietnamese residents for a 70 year lifetime, and exposure to no other source of PCDDs and PCDFs.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch012

from the south of Vietnam the factors of fold-excess intake are 1.5 to 150 (age and weight corrected). For the Vietnam population in the north, these factors are much lower, .03 to 2.2.

Discussion

Although the neonatal body burden of lipid-soluble exogenous chemicals is likely much less than that of the mother, the transfer of these contaminants to milk is likely to be very efficient due to the high proportion of milk fat. Breast milk is considered the ideal food for a certain, but not fully characterized, time interval after birth of the infant. Against the beneficial aspects of human breast milk must be weighed the risks posed by the chemicals, in this case the chlorinated dioxins and dibenzofurans. This may be especially true for populations known to be heavily contaminated with these compounds. In animal models, these chemicals have been shown to increase the risk of developing certain tumors, immune deficiency, adverse reproductive effects including congenital malformations and fetal lethality, hepatic damage, and gastrointestinal and genitourinary system epithelial cell disorders. While it is clear that some toxic effects have been observed in humans exposed to the dioxins, an unequivocal effect on more subtle and/or longer term parameters such as cancer development and alterations of immune function has not yet been conclusively demonstrated. This may reflect unusual, but certainly not unique, species differences between human and other mammalian species. Alternatively, this may be a reflection of inadequacies of epidemiological design and analysis. If dioxins do cause human health effects at the levels seen in industrial countries or in the south of Vietnam, clinical and epidemiologic studies in Vietnam, with its extreme levels of dioxins in human tissues, high in the south and low in the north, may more readily reveal this than elsewhere.

We have used the human tissue levels of PCDDs and PCDFs in the north of Vietnam as a reference value, believing that this area has had little environmental contamination from synthetic, industrial, and agricultural chemicals. The United States, Canada, and Europe represent highly industrialized areas with a substantial use, disposal, and combustion of a variety of synthetic, especially halogenated chemicals, The south of Vietnam, because of the herbicides used for military purposes during the 1960's and 1970's, has had a significant contamination with 2,3,7,8-TCDD as well as other PCDD and PCDF isomers.

Our calculations suggest that levels of these contaminants in breast milk from individuals from the south of Vietnam may exceed recommended standards currently employed by governmental agencies in the United States. Although concentrations of these compounds expressed as TCDD equivalents in North American populations are lower as compared to the south of Vietnam, nursing infants may be consuming levels which are near or above these recommended values. It must be noted that these calculations are based on mean tissue values for which there are wide ranges of variation. We have also made necessary assumptions regarding infant weight, duration of nursing, the average amount of milk consumed, and for the gastrointestinal absorption of these isomers, all of which may or may not be relevant for individual situations. However, we have also assumed that breast milk represents the only lifetime source of exposure to these environmental

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch012

contaminants. This latter assumption appears unlikely given the use, disposal, and combustion of chemicals which may lead to the continued input of these specific toxins into the environment.

Conclusions

The findings of the chlorinated dibenzo-p-dioxin and dibenzofuran isomers in persons residing in contaminated areas and both measured and calculated levels of these compounds in breast milk in the United States presents a possible cause for concern regarding health effects in the nursing infant. Further study is indicated to determine if there is any real clinical risk at the levels found.

Breast fed infants represent a population with daily exposure to dioxins and related chemicals. This population may have an increased theoretical risk for PCDD and PCDF induced cancer and other dioxin induced illnesses. This is especially true for contaminated populations in the south of Vietnam. However, the theoretical risk is based on an extrapolation from animal models and varies considerably on the model chosen. We do not know whether there will actually be an increased cancer incidence at these levels of contamination.

Curtailment of breast feeding is not recommended at this time. However, infant populations consuming elevated levels of the chlorinated dioxins and related chemicals in breast milk (especially in the south of Vietnam) should be followed for longer term effects of dioxin exposure.

Acknowledgments

The authors gratefully acknowledge the use of unpublished data from Dr. J.J. Ryan (17), generously provided for this manuscript.

Financial assistance from the Reynolds Foundation, the Samuel Rubin Foundation and the CS Fund for the work of one of us (A.S.) is gratefully acknowledged.

Literature Cited

1. Schecter, A.J.; Ryan, J.J.; Gitlitz, G. In "Chlorinated Dioxins and Dibenzofurans in Perspective"; Rappe, C.; Choudhary, G.; Keith L., Eds.; Lewis Publishers Inc.: Chelsea, Michigan, 1986; pp. 51-65.
2. Schecter, A.J.; Ryan, J.J.; Gross, M.; Weerasinghe, N.C.A.; Constable, J. In "Chlorinated Dioxins and Dibenzofurans in Perspective"; Rappe, C.; Choudhary, G.; Keith, L., Eds.; Lewis Publishers Inc.: Chelsea, Michigan, 1986; pp. 35-50.
3. Schecter, A.J.; Ryan, J.J.; Constable, J. Chemosphere 1986, 15, 1613-20.
4. "Health Assessment Document for Polychlorinated Dibenzo-p-dioxins," United States Environmental Protection Agency, EPA/600/8-84/014F, 1985.
5. Kimbrough, R.D.; Falk, H.; Stehr, P. J. Toxicol. Environ. Hlth. 1984, 14, 47-93.
6. Morbidity and Mortality Weekly Report 1984, 33, 25-7.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch012

7. Murray, F.J.; Smith, F.A.; Nitschke, K.D.; Humiston, C.G; Kociba, R.J.; Schwetz, B.A. Toxicol. Appl. Pharmocol. 1979, 50, 241-51.
8. Nisbet, I.C.T.; Paxton, M.B. Am. Stat. Vol. 1982, 36, 290-8.
9. "Ambient Water Quality Criteria for 2,3,7,8-Tetrachlorodibenzo-p-dioxin," United States Environmental Protection Agency, EPA/440/5-84-007, 1984.
10. Poland, A.; Palen, D.; Glover, E. Nature 1982, 300, 271-3.
11. Pitot, H.C.; Goldsworthy, T.; Campbell, H.A.; Poland, A. Cancer Res. 1980, 40, 3616-20.
12. International Commission on Radiological Protection in "Report on the Task Group on Reference Man"; Pergamon Press: New York, 1975.
13. Butte, N.F.; Garza, C.; Stuff, J.E.; Smith, E.O.; Nichols, B.L. Am. J. Clin, Nutr. 1984, 39, 296-306.
14. Whitehead, R.G.; Paul, A.A. Lancet 1981, 2, 161-3.
15. Hofvander, Y.; Hagman, U.; Hillervik, C.; Sjolin, S. Acta Ped. Scand. 1982, 71, 953-8.
16. Stanley, J.S.; Boggess, K.E.; Onstot, J.; Sack, T.M.; Remmers, J.C.; Breen, J.; Kutz, F.W.; Carra, J.; Robinson, P.; Mack, G.A. Chemosphere 1986, 15, 1605-12.
17. Ryan, J.J. Report of a World Health Organization Planning Meeting on PCBs, PCDDs and PCDFs in Human Milk, Norway, 1986.
18. Graham, M.; Hileman, F.D.; Orth, R.G.; Wendling, J.M.; Wilson, J.D. Chemosphere 1986, 15, 1595-600.
19. Patterson, D.G.; Hoffman, R.E.; Needham, L.L.; Roberts, D.W.; Bagby, J.R.; Pirkle, J.L.; Falk, H.; Sampson, E.J.; Houk, V.N. JAMA, 1986, 256, 2683-6.
20. Wolff, M.S. Am. J. Ind. Med. 1983, 4, 259-81.
21. Rappe, C.; Bergqvist, P-A.; Hanson, M.; Kjeller, L-O.; Lindstrom, G.; Marklund, S.; Nygren, M. In "Biological Mechanisms of Dioxin Action"; Poland, A.; Kimbrough, R.D., Eds.; BANBURY REPORT 18, Cold Spring Harbor Laboratory; Cold Spring Harbor, 1986; pp. 17-26.
22. Rappe, C.; Nygren, M.; Lindstrom, G.; Hansson, M. Chemosphere 1986, 15, 1635-40.
23. Tarkowski, S.; Yrjanheikki, E. Chemosphere 1986, 15, 1641-8.
24. Barnes, D.G.; Bellin, J.; Cleverly, D. Chemosphere 1986, 15, 1895-903.
25. Eadon, G. et al., New York State Department of Health, 1982.
26. Gravitz, N. et al., California Department of Health Services, 1983.
27. Baughman, R.W. Ph.D. Thesis, Harvard University, Cambridge, MA, 1974.

RECEIVED February 24, 1987

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch012

Chapter 13

Uncertainties in Dioxin Risk Assessment

Vernon N. Houk

Center for Environmental Health, Centers for Disease Control, and Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA 30333

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch013

An evaluation of human health hazards posed by dioxin-contaminated soil in certain areas of Missouri illustrates specific uncertainties in dioxin risk assessment. These uncertainties include the relationship of studies in animals to effects in humans, the level and extent of contamination in the soil, the level of exposure to humans, and the character of the dose-response curve. Uncertainties in assessing risks posed by toxic substances in general are viewed from the standpoint of a 1985 report prepared by the Task Force on Risk Assessment and Risk Management for the Secretary, Department of Health and Human Services. The Task Force listed nine commonly used assumptions--which may also be called uncertainties.

In 1981, the Environmental Protection Agency (EPA), through its regional office in Kansas City, Missouri, and the State of Missouri, asked the Centers for Disease Control (CDC) to evaluate the human health hazards posed by dioxin-contaminated soil in certain areas of Missouri. Industrial wastes containing dioxin (2,3,7,8-tetrachlorodibenzodioxin or dioxin) had been mixed with salvage oil, and in 1971 the mixture was sprayed on riding arenas and dirt roads in eastern Missouri to control dust. An outbreak of poisoning in horses and other animals followed. In 1974, CDC established that the riding arenas had been contaminated with dioxin. Recently, however, investigators have found that the soil in several other areas--many of them residential--was also contaminated. Concentrations in the more recently examined soil samples have ranged from less than 1 to more than 1,000 parts per billion--measured in micrograms of dioxin per kilogram of soil.

Details of the Center for Environmental Health's assessment of the risk to human health of dioxin-contaminated soil in residential areas are in an article in the Journal of Toxicology and Environmental Health (1). A shorter version appeared in the CDC

This chapter not subject to U.S. copyright.
Published 1987, American Chemical Society

Morbidity and Mortality Weekly Report (2). The uncertainties in the Missouri study are covered in these two reports.

Missouri Study: Uncertainties

Since no information was available on the specific doses of dioxin that caused or did not cause health effects in humans, we reviewed the data from studies in animals. These studies show that some animal species are more susceptible to the toxic effects of dioxin than others, but we do not know which species responds most like humans.

Some scientists have claimed that humans must not be very susceptible to the toxic effects of dioxin because, despite all the exposures in the workplace, the main health effect in humans is chloracne. Although worker populations have had most of the heavy exposures, they may not fully reflect the risks for the general population. In estimating the risks for the general population, we must consider other groups--children, women of childbearing age, the aged, and the infirm.

Another uncertainty is the effect of long-term exposure. Many short-term tests of dioxin's acute effects have been conducted in animals, but at the low levels of contamination in Missouri, acute effects are not as important as chronic effects that might follow long-term daily exposure. Studies that provide information on the chronic effects of dioxin include a few conducted in rodents to determine whether or not dioxin is a carcinogen. We used the results of these studies to calculate a dose that might represent a reasonable risk of exposure for humans.

The estimate of how much dioxin humans take up after exposure to contaminated soil was also based on uncertainties, that is, on assumptions that have not been verified. We estimated how much dioxin people exposed to contaminated soil adjacent to their homes would absorb via the skin or gastrointestinal or respiratory tracts. We assumed that each daily dermal contact with soil would, in most instances, result in a 1 percent uptake of dioxin from 1 gram of soil. We also estimated how frequently adults and older children would come in direct contact with soil.

As for small children, we assumed that when they played outside they might eat as much as 10 grams of soil per day, although they were more likely to eat 1 gram. We assumed that 30 percent of the dioxin bound to the ingested soil might be absorbed from the gastrointestinal tract, and then we estimated how often a child would play outside and for how much of his or her lifetime the child would ingest soil.

Since we were evaluating residential areas with abundant vegetation, and consequently not much dust, we did not think that inhaling dioxin-contaminated dust would be a significant problem.

Another uncertainty was the level and extent of contamination. An entire area is not contaminated at the highest level found anywhere in the area. Furthermore, dioxin contamination is more likely to be limited to a percentage of the total area. We calculated what the dose would be if only 10 or 20 percent of the soil in an area were contaminated. In addition, the dioxin was applied in an oil mixture, so it might not have been bound to the soil as tightly as it would have been had it not been applied in the

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch013

mixture. Recent studies have shown that without the oil, the dioxin would have been bound much tighter.

No one knows whether the dose-response curve for carcinogens like dioxin is linear--that is, that the response is directly proportional to the dose. Nevertheless, we assumed a linear relationship, and this could have caused us to overestimate the risk. Further, as suggested above, we assumed that a chemical shown to be a carcinogen in animals is also a carcinogen in humans.

Despite these uncertainties, in 1983 CDC advised Missouri that, in two residential areas, levels above 1 part per billion of dioxin per kilogram of soil could result in an unreasonable risk to human health. Later on June 28, 1983, a group of outside consultants reviewed this assessment and concurred.

Task Force Report: Uncertainties

The uncertainties in any risk assessment were addressed in the April 1985 report to the Secretary, Department of Health and Human Services (3). In January 1985, Secretary Margaret Heckler asked Dr. James O. Mason, Chairman of the Executive Committee of the Department's Committee to Coordinate Environmental and Related Programs (CCERP), to analyze the Department's policies and practices on assessing and managing risks posed by toxic substances. As a result, the Task Force on Risk Assessment and Risk Management was formed. It was made up of senior scientists from the Centers for Disease Control, the Food and Drug Administration, and the National Institutes of Health. A group established by CCERP's Executive Committee guided the Task Force, and I was Chairman of this group.

The final product of any risk assessment generally produces numbers that appear to have precision, but the Task Force emphasized that a risk assessor should clearly define the differences in the elements that go into the process as scientific fact, consensus, assumption, and "science policy"--or the agency decision on how it is going to handle controversial issues. The Task Force listed nine commonly used assumptions--which may also be called uncertainties: (1) When human data are not adequate, adverse effects in experimental animals are regarded as indicative of adverse effects in humans; (2) results obtained with dose-response models can be extrapolated outside the range of experimental observations to yield estimates or estimated upper bounds on low-dose risk; (3) when an appropriate standardized dosage scale is used, observed experimental results can be extrapolated across species; (4) there is no threshold for the production of cancer, but threshold effects may apply for other toxicologic outcomes; (5) when dose rates are not constant, average doses give a reasonable measure of exposure; (6) in the absence of pharmacokinetic data, the effective target dose is assumed to be proportional to the administered dose; (7) risks from many exposures and from many sources of exposure to the same chemical are usually assumed to be additive; (8) in the absence of evidence to the contrary and regardless of the route of exposure, 100 percent absorption across species is assumed; and (9) results associated with a specific route of exposure are potentially relevant for other routes of exposure.

The Task Force cautioned that in any risk assessment, an attempt should be made to clearly identify all of the main

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch013

assumptions made and to indicate their probable effect on the risk assessment.

As the Task Force stated, risk assessment is not a static process and its "evolutionary nature is, perhaps, best characterized by the increasing emphasis . . . on identifying and reducing the uncertainties associated with the process." Various organizational units in the Department are attempting to improve or expand data bases on human exposures and health effects and to further characterize the toxicologic profiles of hazardous agents. Some units are conducting basic and applied research in toxicology and other fields that bear on some of the uncertainties.

The Task Force also developed, in concept, a problem-solving approach to risk management that provides a framework for overcoming difficulties by analyzing the risk assessment, analyzing options, promoting understanding and acceptance of risk-management decisions, and evaluating the effectiveness of the options chosen. This is a framework through which policy makers can assess information, determine risks faced by the public, and allow for the democratic process to decide the acceptability of each risk on an incremental basis. In this approach, each risk management action can be seen as a solution to a problem--a solution from which we can learn.

Conclusion

The uncertainties in dioxin risk assessment in Missouri are many--and there are many in the risk assessment and risk management of any toxic substance anywhere. But, we are working to reduce these uncertainties and, as this is done, our assessments of risks and our management of risks should continue to improve.

Literature Cited

1. Kimbrough, R. D.; Falk, H.; Stehr, P. J. Toxicol. Environ. Health. 1984, 14, 47-93.
2. Centers for Disease Control. MMWR 1984, 33, 25-27.
3. Risk Assessment and Risk Management of Toxic Substances: A Report to the Secretary, Department of Health and Human Services (DHHS), from the Executive Committee, DHHS Committee to Coordinate Environmental and Related Programs (CCERP): April 1985; DHHS [Centers for Disease Control]: [Atlanta, GA], 1985.

RECEIVED December 15, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch013

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

Chapter 14

Assessing the Potential Human Health Hazards of Dioxin-Contaminated Soil

D. J. Paustenbach, H. P. Shu, and F. J. Murray

Environmental Health and Safety, Syntex (USA), Inc., 3401 Hillview Avenue, Palo Alto, CA 94303

Regulatory agencies worldwide are currently considering environmental standards for 2,3,7,8-tetrachlorodibenzo-p-dioxin (dioxin, TCDD). This paper assesses the validity of assumptions which underlie the proposed approaches to setting limits for TCDD in the soil of residential and industrial sites. This paper also offers alternatives to these assumptions which are more justifiable scientifically, which profoundly affect the conclusions of the risk assessment, and which alter the magnitude of the recommended limits. Specifically, these assumptions concern 1) the quantities of soil typically ingested by children, 2) TCDD's nongenotoxicity, 3) the extent of dermal contact with soil, 4) the concentration of airborne soil particles, 5) dioxin's bioavailability in soil, 6) extrapolation of the dose response curve, and 7) appropriate risk criteria for small exposed populations. Two case studies are presented which illustrate the quantitative effect of these assumptions on the exposure estimates. Non-U.S. regulatory agencies which have incorporated TCDD's nongenotoxicity in their approach have estimated the virtually safe dose (VSD) or acceptable daily dose (ADI) for TCDD at up to 10 pg/kg/day (10,000 fg/kg/day). The approaches of these agencies are compared and contrasted with the method used by the United States EPA whose risk estimates are higher and whose VSD is approximately 1,000-fold lower. An alternative analysis of the cancer data, which is more valid scientifically than approaches which have been based on regulatory policy, estimates a VSD of 130 pg/kg/day. In light of these many considerations, in residential areas a soil concentration of TCDD considerably in excess of 1 ppb should be acceptable. Soil concentrations in excess of 100 ppb in non-residental settings should amply protect the environment and public health.

0097-6156/87/0338-0178$10.00/0
© 1987 American Chemical Society

Public and regulatory attention in the United States and Europe has focused on setting limits for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD or dioxin) in soil, water, air, fly ash, fish and other foods. In 1982, the U.S. Centers for Disease Control (CDC) developed a risk assessment to evaluate the hazard posed by soil contaminated with low levels of dioxin. The closing sentence in the abstract of the CDC publication summarizes its principal conclusion: "One part per billion of 2,3,7,8-TCDD in soil is a reasonable level at which to ***begin consideration*** of action to limit human exposure for contaminated soil." (emphasis added) (1) This manuscript discusses various assumptions which underlie the assessments by CDC and the U.S. Environmental Protection Agency (EPA) (2) and recommends refinement of the assumptions.

Kimbrough et al noted that to apply accurately the methodology they suggested for assessing risk, at least seven caveats should be heeded (1,3); namely, [a] the 1 ppb level for surface soil was intended to be only a guideline, developed for only one set of conditions, not a standard to be applied anywhere TCDD is present in soil; [b] this guideline would not apply to industrial sites; [c] the 1 ppb guideline assumes that 100% of the soil surface area is contaminated with TCDD; [d] the recommendation assumes that persons would spend a lifetime (70 years) in the contaminated area; [e] one must consider the ease of human access to the contaminated area; [f] dietary uptake should be considered, when appropriate; and [g] whenever new or better data become available, these should be incorporated into the assessment methodology. The EPA described similar caveats in its risk assessment for dioxin contaminated soil (2).

Despite CDC's caveats, regulatory agencies within the United States have in many cases adopted the CDC's 1 ppb guideline as a generic standard for clean-up ***regardless*** of the circumstances at the site. For example, EPA has functionally implemented a 1 ppb standard for clean-up at most of its remediation sites within Missouri, regardless of the potential for human exposure (4,5). It is critically important that regulatory agencies responsible for establishing ***clean-up*** levels be familiar with the underpinnings of the methods and assumptions used in any risk assessment of TCDD contaminated soil. The primary reason is that the cost of remediation varies dramatically with the degree of clean-up and excessive remediation would not make optimal use of America's limited financial resources. For example, the cost of removing and disposing of soil containing more than 1 ppb at the Castlewood site in Missouri, was estimated to be $17,000,000 (Figure 1). To illustrate the dramatic effect of the criteria for clean-up on cost, if 10 ppb were the clean-up level, the cost would drop to $6,000,000, and minimal action would be required at 100 ppb (using EPA estimates for clean-up costs of $1500 per cubic yard of soil excavated and incinerated). Other sites, such as Times Beach, Missouri show an even more dramatic relationship between the cost of clean-up and the degree of remediation.

The following discussion addresses both the CDC and, to a lesser degree, the EPA risk assessments. We suggest specific alternative assumptions which appear more consistent with the

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

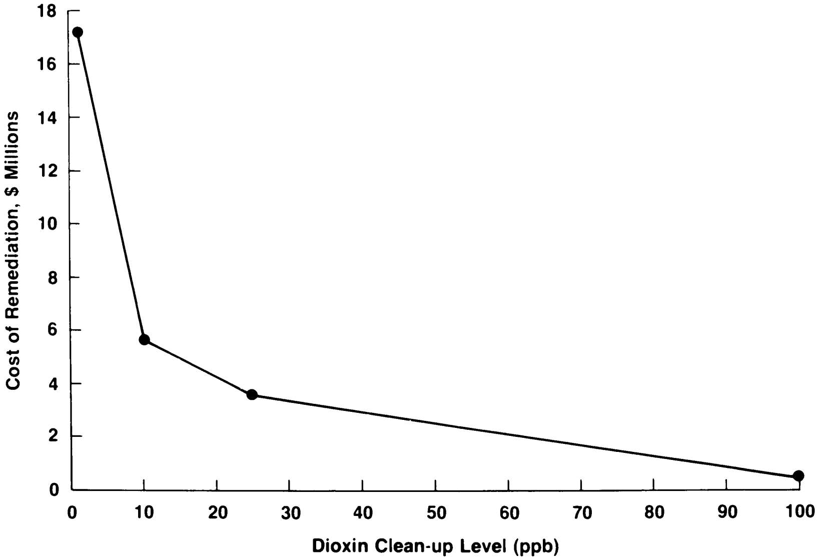


Figure 1: Estimated cost of soil removal and destruction (clean-up) for various target soil contamination levels at the Castlewood site in Missouri. Plot is based on EPA estimates of the number of yards of soil contaminated at various concentrations of dioxin and their estimated cost for incinerating. Reproduced with permission from ref. 108. Copyright 1986, Academic Press.

current scientific data. Although there are many factors to consider when evaluating the health hazards posed by contaminated soil, this paper will address a few of the most critical ones, including exposure from soil ingestion, inhalation and dermal contact, bioavailability of soil-bound dioxin via ingestion, dermal contact, and inhalation, dioxin distribution at a site, human access to residential vs industrial sites, genotoxicity, the dose-response extrapolation, and the appropriate risk criteria for situations where only small numbers of persons are exposed.

EXPOSURE FROM INGESTION OF SOIL

Exposure is clearly one of the most important aspects of a risk assessment. In CDC's assessment, its estimates indicated that the primary route of exposure was soil ingestion (Table I). CDC predicted that about 95% of the average lifetime uptake of TCDD would occur as a result of soil ingestion; about 3% of the lifetime dose would be absorbed through the skin as a result of contact with contaminated soil (associated with gardening and poor hygiene); and no more than 2% of the total dose would be due to inhalation of TCDD-contaminated dust. Based on the information presented in CDC's manuscript, it was implied that a daily uptake of about 636.5 fg/kg/day of TCDD did not pose a health hazard since, using CDC's assumptions, this is the estimated amount which could potentially be absorbed (lifetime average) by persons living in communities which contain soil contaminated with 1 ppb of TCDD. CDC's figure of 636.5 fg/kg/day was selected based on consideration of both the upper bounds and fitted model values on the cancer risks estimated by mathematical models used for extrapolation of the data obtained in two bioassays of dioxin (6,7) as discussed by Portier *et al* (8).

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

TABLE I: CDC Estimates of TCDD Uptake by Route of Exposure (1 ppb in soil)

Route	Avg. daily dose (fg/kg/day)	% Lifetime uptake
Ingestion of soil	606	95
Dermal uptake of soil	20	3
Uptake of soil inhaled	10	2

In its calculations, CDC assumed that as much as 10 grams per day of soil would be ingested by children aged 1.5 to 3.5 years and that during other periods ingestion would be much less, depending on age (Table II). Persons older than 5 years were assumed to ingest 100 mg/day through incidental ingestion (1).

If these assumptions are used, the resulting calculations indicate that about 80% of the entire lifetime dose occurs during the first 5 years of life.

TABLE II: Ingestion of Dirt* (CDC Assumption)

Age group	Soil ingested	% Lifetime uptake
0-9 Months	0 mg/day	0
9-18 Months	1000 mg/day	2.6
1.5-3.5 Years	10,000 mg/day	70.0
3.5-5 Years	1000 mg/day	5.2
5-70 Years	100 mg/day	22.6

*Adjusted for seasonal variations

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

In light of the critical role which soil ingestion plays when estimating human exposure to contaminated dirt, a survey of the literature was undertaken to identify the typical amount consumed by children and adults. Walter and co-workers (9) have estimated that a normal child typically ingests very small quantities of dust or dirt between the ages of 0-2, the largest quantities between ages 2-7, and nearly insignificant amounts thereafter. In the classic text by Cooper (10), she notes that the desire of children to eat dirt or place inedible objects in their mouths "becomes established in the second year of life and has disappeared more or less spontaneously by the age of four to five years." A study by Charney et al (11) indicated that mouthing tends to begin at about 18 months and continue through 72 months depending on several factors such as nutritional and economic status as well as race. Work by Sayre et al (12) also showed that the ages 2-6 years are the ones of primary concern.

An important subtlety that is often lost in the scientific literature is that mouthing tendencies and the ingestion of very small quantities of dirt associated with poor hygiene should not be called pica. Children who intentionally eat dirt, plaster or paint chips and, as a consequence, have developed chronic health problems can be said to suffer from the disease known as pica (10). If the craving is for dirt alone, this disease is known as geophasia. Further, the incidence of pica in the general population has often been overstated because some of the best studies were conducted in children who already suffered from lead poisoning due, in part, to their strong desire to eat dirt and other small objects. In such populations, the incidence of pica has been reported at about 20% (10,16). In contrast, in the general population the incidence of pica is probably in the range of 1-3% (11) while geophasia is more rare.

Duggan and Williams (13) have summarized the literature on the amount of lead ingested through dust and dirt. In their opinion, a quantity of 50 μg of lead was the best estimate for

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

daily ingestion by children from dust. Assuming, on the high side, an average lead concentration of 1000 ppm, this would indicate an ingestion of 50 mg/day of soil and dust per day. Lepow and co-workers (14,15) estimated a rate of ingestion equal to 100 mg/day (specifically, 10 mg ingested 10 times a day). Barltrop (16) also estimated that the potential uptake of soils and dusts by a toddler is about 100 mg/day. A report by the National Research Council which addressed the hazards of lead (17) suggested a figure of 40 mg/day. Day et al (18) also suggested a figure of 100 mg/day (based on eating soiled candy) and Bryce-Smith (19) estimated 33 mg/day. In their document addressing lead in air, EPA (20) assumed that a child ate 50 mg/day of household dust, 40 mg/day of street dust, and 10 mg/day of dust derived from their parents' clothing (i.e., a total of 100 mg/day).

More recently, Hawley (21) reviewed the available literature on soil ingestion and concluded that uptake was approximately 100 mg/day but he developed a more complex lifetime exposure schedule than had been proposed by others (Table III). Bellinger et al (22) have suggested a figure of 20 mg/day. Binder et al (23) conducted a pilot study which employed a rigorous experimental approach involving the analysis of trace elements in children's stool samples. Their data indicated that children 1 to 3 years of age ingest about 180 mg/day of soil (geometric mean) based on the quantity of silicon, aluminum and titanium found in the feces. The limitations of this study and the difficulties encountered in the interpretation of the titanium data have been reviewed by Clement Associates (24).

TABLE III: Other Estimates for Ingestion of Soil by Humans

Age group (years)	Lepow *et al.* (14) [a] (mg/day)	Duggan and Williams (13) (mg/day)	Baltrop (14) (mg/day)	Hawley (21) (mg/day)	Van Wijnen *et al.* (25) (mg/day)
0-2	— [b]	—	—	Negligible	—
2-6	100	50	100	90	56
6-18	—	—	—	21	—
18-70	—	—	—	57	—

[a] Used in EPA TCDD risk assessment.
[b] —Indicates the researchers did not discuss these age groups.

In a very recent study, van Wijnen et al (25) reported the results of a study of 2-4 year old children wherein the feces were analyzed for titanium, aluminum, and acid soluble residue (air). The results showed that the data were approximately normally distributed. Estimates of 105 mg/day for nursery school children and 49 mg/day for the hospitalized children were obtained. Even with the limited number of samples in the study ($n = 24$), the difference between the two populations was significant ($p < 0.01$). If the value for the hospitalized

In Solving Hazardous Waste Problems; Exner, J.;
ACS Symposium Series; American Chemical Society: Washington, DC, 1987.

children is assumed to be the background due to intake of these substances from non-soil sources (e.g., diet and toothpastes), the estimated average soil ingestion of this group of nursery school children would be 56 mg/day. This value is in the lower range of the estimates in the literature and supports the use of 100 mg/day as a reasonable daily average uptake of soil by toddlers (ages 2-4 or ages 1.5 - 3.5).

For the majority of persons beyond the age of 5-6, the daily uptake of dirt due to intentional ingestion should be quite low. One route by which adults ingest dirt is presumably fruits and vegetables; and nearly all of it is due to leafy vegetables (20,26). Another source of ingestion is poor personal hygiene (31). Investigations at nuclear weapons trials have shown that particles which exceed 45 um are seldom retained on leaves (27). In addition, the superficial contamination by the smaller particles is readily lost from leaves; either by mechanical processes or rain, and certainly by washing (26,29).

It has been estimated that the deposition rate of dust in rural environments is about 0.008 ug/cm^2-day (20) assuming that rural dust contains about 500 ug/g of lead (the substance for which these data were obtained). EPA (20) also estimated that even at relatively high air concentrations (i.e., 0.45 mg/m^3 total dust), it is unlikely that surface deposition alone can account for more than 1.0-2.5 mg dust per gram of lettuce (2-5 ug/g lead) on the surface of lettuce during a 21-day growing period. Based on EPA upper worst case estimates for lead uptake via gardens (100 µg lead/day), the corresponding value for ingestion of dust would be 10 mg/day (at 1000 µg/g lead). These data suggest that the daily ingestion of dirt and dust by adults is unlikely to exceed 0-5 mg/day even if all of the 137 g of leafy and root vegetables, sweet corn and potatoes consumed by adult males each day were replaced by family garden products. This figure is much lower than the 100 mg/day figure used by CDC to describe average daily soil ingestion by adolescents and adults (ages 6-70). The actual uptake by adults from vegetables should be virtually zero since this estimate assumes that all dust is contaminated with dioxin, that persons don't wash the vegetables, that photodegradation does not occur, and that garden vegetables are eaten throughout the year rather than only during the growing season.

When all this published information on soil ingestion is considered, the data indicate that a consensus estimate for soil ingestion by children (ages 1 1/2 - 3 1/2) is about 100 mg/day. This figure was adopted by the EPA in its risk assessment (2), and in the Superfund Health Assessment Manual (30). The likely average uptake of contaminated soil by adults should also be much less than that estimated by CDC, perhaps as low as 0-5 mg/day. Most persons wash their vegetables before ingestion and the hygienic practices of most persons are not so poor as to make this route of uptake very significant. The impact of these assumptions regarding soil ingestion by children and adults on the risk estimates is dramatic. Depending on the site on which a risk assessment is conducted, as will be shown later in this

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

paper, the use of 100 mg/day for those aged 2-4, rather than 10,000 mg/day, and 0-5 mg/day rather than 100 mg/day for ages 6-70 years, will significantly change the estimates of risk.

EXPOSURE FROM INHALATION

Although some persons have been concerned about inhaling dioxin, either as a vapor or as a contaminant of ambient dust, both the EPA, CDC and other assessments have demonstrated that inhalation does not constitute a significant route of entry for TCDD. If, as assumed by CDC, the average air concentration of total suspended particulates (TSP) is 0.14 mg/m^3 and 100% of this amount (by weight) is respirable, then the average daily uptake over a lifetime is estimated at 10 fg/kg/day - roughly 2% of the amount taken up by ingestion, according to the CDC calculations (Table 1).

Inhalation is actually a less important route of entry than assumed by CDC, if other data are considered. For example, the EPA has estimated that the average concentration of total suspended particulates (TSP) in Missouri is about one-half the level assumed by CDC, or about 0.070 mg/m^3 (32,33). Secondly, only a fraction (about 30%) of the total amount of airborne dust is respirable (less than 10 µm aerodynamic diameter) (32). The percentage of TSP which is respirable has been estimated by EPA to be no more than 50% (2). Also, the assumption that all of the airborne particles are derived from dioxin-contaminated soil is overly conservative. Actually, about 50% of the inhaleable particles are respirable and the remainder are between 10 and 50 µm. Further, about 83% of the non-respirable particles are from crustal material (e.g., soil) and only 47% of the respirable particles are from soil (32). Larger studies conducted by the U.S. EPA have suggested that the portion of inhaleable dust due to soil can often be much less (34).

EXPOSURE FROM DERMAL CONTACT

To estimate daily TCDD uptake via the skin, CDC assumed that dermal exposures would follow "an age-dependent pattern of deposition similar to soil ingestion" as shown in Table IV (1). There are several subsequent assumptions which would over estimate the degree of dermal exposure. CDC assumed that dirt would remain on the hand for a period long enough to bring about 1% absorption (1) - the percent absorption determined in rats exposed for 24 hours (35). A 24-hr duration is almost certainly longer than what is likely for humans under normal conditions. Secondly, human skin has generally been shown, for a diverse class of chemicals, to be less permeable to xenobiotics than the skin of rabbits and rats (36-38).

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

TABLE IV: Amount of Soil Deposited on Skin (CDC Assumption)

Age group	Soil on skin (day)
0-9 Months	0 g
9-18 Months	1 g
1.5-3.5 Years	10 g
3.5-5 Years	1 g
5-70 Years	100 mg

In both assessments, CDC and EPA assumed that the likelihood for dermal exposure would be affected by weather conditions, and consequently, made adjustments. In the CDC calculations, it appears that CDC assumed that persons would come into contact with soil or garden for about 180 days per year for 64 years [70 minus 6]. This estimate is almost certain to overestimate exposure since [a] not everyone in the community gardens; [b] many persons wear gloves when working intimately with dirt; [c] gardeners work directly with the soil primarily during planting and weeding; and [d] most people do not garden each day.

EPA, in its risk assessment, used an alternative set of assumptions which, because they are based, in part, on actual field investigations, seem more realistic than CDC's assumption in Table IV. A study by Roels et al (38) has indicated that about 0.5 mg of soil per cm^2 of skin adheres to a child's hand after playing in and around the home. This is similar to that reported by Day et al (18) who observed that 5-50 mg of dirt transferred from a child's hand to a sticky sweet. Brunekreef et al (39) found similar results. Using table values for skin surface area (40), uptake can be estimated by multiplying this value by the 1% absorption rate and the amount of dirt on the skin. Depending on the conditions of exposure, this alternative approach - which could be based on skin surface area, duration of exposure (time), and the amount of dirt on the skin - could predict a markedly different, often lesser, degree of dermal uptake than CDC's assumptions.

BIOAVAILABILITY: DERMAL ABSORPTION

Bioavailability is an important parameter in any risk assessment of dioxin. Throughout this discussion, bioavailability will be used to describe the percentage of TCDD in soil which is absorbed by humans (as suggested in animal studies) following exposure via inhalation, ingestion or dermal contact. There are a number of parameters which are likely to influence the degree of bioavailability of dioxin when it is on soil including aging (time following contamination), soil type (e.g., silt, clay and sand), co-contaminants (e.g., oil and other organics), and the

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

dioxin concentration on the soil. Poiger and Schlatter (35) have conducted the only published study on dermal bioavailability. They dosed rats dermally with laboratory contaminated soil and observed that as the dose increased, the liver concentration of TCDD increased from 0.05 to 2.2%. The authors did not estimate a value for dermal bioavailability. On the basis of this study, Kimbrough and co-workers estimated a dermal bioavailability of 1% for humans (1). The use of a 1% dermal absorption factor (bioavailability) almost surely overestimates the actual uptake of TCDD on soil through human skin since investigators in the dermal field generally agree that rodent skin is approximately 10 times more permeable than human skin. As discussed in the next section, dermal bioavailability (like oral bioavailability) is also likely to decrease with the "age" of the soil. (41,42)

BIOAVAILABILITY: INGESTION

Poiger and Schlatter (35) published the first study on this topic. They dosed rats orally with laboratory prepared TCDD contaminated soil and monitored the % of administered dose in the liver. Their data suggest that as the time of contact between the soil and TCDD (known as aging) increased, the oral bioavailability decreased. Other investigators have also observed that aging may decrease bioavailability (41,42).

McConnell et al. (43) studied Missouri soil contaminated with TCDD. They looked at the liver concentration of TCDD in the guinea pig and rat, and arylhydrocarbon hydroxylase (AHH) induction in the rat, following soil ingestion. They concluded that TCDD absorption from soil by test animals is highly efficient, but that they had difficulty in arriving at an exact percentage for bioavailability. In the CDC assessment, Kimbrough et al. (1) used a 30% bioavailability value and cited McConnell et al. (43) as the reference. Lucier et al. (44) republished some of the original data from McConnell et al. (43) and concluded the oral bioavailability was 50%. However, the data of Lucier et al. (44), suggested the bioavailability was dose dependent - 24% at 1 µg/kg and 50% at 5 µg/kg TCDD. Lucier et al. estimated oral bioavailability by comparing the liver TCDD concentration of control rats dosed with TCDD in corn oil with experimental rats dosed with TCDD on Missouri soil.

In a 1985 abstract, Umbreit et al. (45) reported bioavailability of less than 0.05% for a New Jersey manufacturing site. This work was subsequently published (46) wherein they reported oral bioavailability of 0.5% for soil at a manufacturing site and 21% for a salvage yard in Newark. In this paper, Umbreit et al attributed an oral bioavailability of 85% to the data presented in the 1984 paper by McConnell et al. Umbreit et al. did not discuss how they defined or calculated bioavailability for their own data or in their interpretation of McConnell et al's data. Work by Umbreit et al (47) has shown that the bioavailability of TCDD in contaminated soil samples from Times Beach and New Jersey can not be reliably assessed using AHH induction due to the presence of co-contaminants.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

Some of the confusion concerning the value for oral bioavailability undoubtedly arises because of the ways investigators have calculated bioavailability. Apparently, some may have used AHH induction as the basis for calculation, while others have used actual liver levels of TCDD. The difficulties in using AHH induction to assess the bioavailability of dioxin-bound soil has been discussed (47). Thus far, no one has used a total material balance, including the amount in all tissues and in excreta, as the basis for comparison; an approach which would yield a much more useful estimate of uptake. Recently, officials of the EPA (48) and CDC (49) cited 85% as the bioavailability of dioxin in soil, based on the available data, in spite of the apparent differences between testing methods and methods for defining bioavailability.

Bonaccorsi et al. (50) have also published a paper on oral bioavailability in the rabbit. They compared liver levels of dioxin 7 days after an oral dose of Seveso soil or a comparable TCDD dose in alcohol. They reported that absorption of soil-bound TCDD from Seveso was 32% that of TCDD in alcohol.

The divergent results on oral bioavailability reported in the literature may occur for several reasons, which will be discussed below. Umbreit and co-workers (46) and Poiger and Schlatter (33) have also offered explanations for the discrepancy. The dioxin concentrations in the soil samples for the various laboratories were similar, but other conditions were quite different. For example, the bolus size (the amount of soil administered to the animal) varied markedly among the studies, as did the amount of TCDD taken up in the liver. The data from the three studies suggest that the larger the quantity of soil (i.e., larger dose of dioxin) given to the animal, the larger percentage of uptake by the liver. There are a number of physiological (e.g., residence time and G.I. motility), biochemical (e.g., liver enzyme induction) and physical (e.g., low concentrations bind more tightly to soil, soil type, co-contaminants) reasons why this might be expected. The guinea pig data suggest that when the amount of soil ingested is low (on a mg/kg basis) - a condition which more closely resembles that seen in children - the percent GI absorption is lower than the 30% figure used by CDC. Due to a lack of experimental data, a similar inference cannot be drawn for the rat.

The level of organic matter in soil may also be an important variable. The New Jersey soil used by Umbreit et al (45) contained a high organic loading in the form of asphalt-like residues, as well as natural organic content. Interestingly, when this soil was stripped of its organic loading, and the dioxin was reapplied, bioavailability approached 23% (45), which supports the hypothesis that increasing amounts of organics in the soil may decrease the bioavailability of TCDD.

In summary, 30% bioavailability of TCDD in soil in the G.I. tract may represent an upper estimate. Given the low concentrations of TCDD usually encountered in the environment and the small daily oral dose anticipated among residents, a figure of 10% may be a reasonable estimate of the average oral

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

bioavailability that might be anticipated for many contaminated sites.

BIOAVAILABILITY: INHALATION

Because TCDD has a very low volatility, TCDD uptake via inhalation is directly related to the concentration of airborne dust due to wind-blown soil. It has been estimated that of all airborne, respirable particulates, only about 30-50% comes from soil, while the rest is apparently due to products of combustion, tire wear and other sources (32). Of the total suspended particulates, usually no more than 50% are respirable (i.e., particles less than 10 um). Of these, about 50% of the respirable particles are deposited in the upper airways and ultimately swallowed while the rest reach the alveoli or are expired. An analysis of CDC's data indicates that CDC assumed that 100% of the TCDD present on all the inhaled particles would be retained and absorbed in the respiratory tract. In contrast, the EPA assessment (2) assumed that only 25% of the inhaled particles would be absorbed in the lower airways since at least 50% of the particles would be non-respirable (especially by weight) and these will be swallowed due to impaction in the throat and only about 50% of the respirable particles would be absorbed. In any assessment, it is important to recognize that of those particles swallowed, no more than 10-30% should be absorbed since they will pass through the G.I. tract (assuming 10-30% oral bioavailability).

EFFECTS OF TCDD DISTRIBUTION AT THE SITE

Kimbrough et al (1) observed that of all the assumptions used in their risk analysis, the "most prominent of these is the assumption of uniform levels of contamination throughout the living space (environment)". CDC was especially sensitive to this issue and generated a very interesting plot which illustrates the relationship between the magnitude of the risk and the percent of the surface area of the land that has been contaminated (Figure 2). It is important to recognize that the degree of human exposure (ingestion, inhalation and dermal) is almost always a function of the TCDD concentration at the soil surface (top 1-2 cm) rather than the concentration at lower depths. This is significant since recent research suggests that within 18 months, nearly all of the TCDD in the top 1/4 cm of soil, and most of it within 1/2 cm, is no longer present due to photodegradation and volatilization (51-53). Photodegradation of TCDD in soil may require the presence of a hydrogen donor, e.g., a solvent or humic soil (52).

RESIDENTIAL VERSUS INDUSTRIAL SITES

Probably the most overlooked of the many caveats in the CDC assessment is the one involving the setting of different clean-up

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

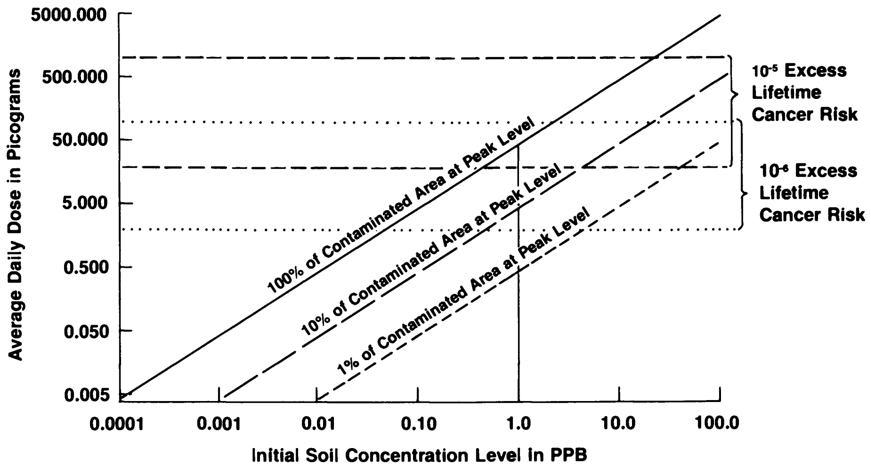


Figure 2: Effect of the degree (prevalence) of dioxin soil contamination at a residential site versus the estimated risk (from Kimbrough et al, 1984). This plot is based on an acceptable daily lifetime dose of 636 fg/kg/day (CDC's best estimate of a dose which would increase the individual cancer risk by 1 in 1,000,000). Reproduced with permission from ref. 108. Copyright 1986, Academic Press.

and health alert guidelines for residential versus industrial sites. As noted in CDC's publication, "in all of these scenarios (factories, farms, residential sites), these decisions must be made on a site-specific basis". The most prominent difference between a residential and an industrial setting, with respect to the hazard to human health, is the lack of access by the public, and, in particular, children to industrial sites. In the absence of such access, at least 95% of the potential exposure to dioxin is eliminated (Table I). In light of the characteristics of industrial sites, the CDC suggested that ***unless*** soil "levels are very high - e.g., above 100 ppb - more extensive remedial action" than paving may not have to be considered (1). The implication is that soil levels at or below 100 ppb at a non-residential site may be acceptable.

APPLICATIONS OF THE CDC METHODOLOGY

As noted in both the CDC and EPA assessments, the process of setting standards for clean-up should be done on a site-specific basis. The task of setting "clean-up levels" and assessing the risk of contaminated soil on a case-by-case basis need not be a process so complex, or cumbersome, as to be impractical for regulatory agencies. Certainly, an algorithm which incorporates all of the important variables could be developed, as illustrated by Hawley (21) and Schaum (2), and these calculations can easily be handled by a desk top computer.

The following case studies illustrate how the CDC methodology could be applied, on a site-specific basis, incorporating some of the data we have described on soil ingestion and bioavailability. These examples illustrate the effect of altering only a few of the critical assumptions in the CDC assessment and the effect of following CDC's advice concerning site-by-site analyses. In developing a comprehensive risk assessment, it is important to acknowledge the uncertainties, as recommended by the National Academy of Science (54). CDC recognized the fragility and uncertainty of many of the assumptions it used and specifically noted that "it must be stressed that the exposure assessments used in estimating risks for carcinogenicity and reproductive health effects contain critical assumptions that are not likely to be actually encountered." Although not addressed in detail in this paper, other critical issues, including the use of classic cancer risk models to estimate TCDDs carcinogenic potency in humans at low doses (despite its lack of genotoxicity) and the incorporation of the generally favorable epidemiology data should be examined in any comprehensive review of a risk assessment of dioxin (55-58).

CASE I (A RESIDENTIAL SITE)

In this example, a hypothetical residential site will be evaluated. For the sake of this discussion, 10% of the soil surface in this community is assumed to be contaminated with 10 ppb TCDD. Altering only three different assumptions in the CDC

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

assessment process, would the analysis suggest that clean-up is needed at this site if only a few of the CDC values were updated?

Using the methods described by the CDC and the EPA, a preliminary evaluation of the need for clean-up at a given site can be readily conducted. In Case I, we followed the CDC assessment procedure but updated a few of their values. Specifically, in these calculations we used: [a] a soil uptake of 100 mg/day (ages 2-6), and negligible soil ingestion for the rest of the lifetime: [b] 10% oral, and 1% dermal bioavailability; [c] quantitative assumptions on dermal exposure, [d] actual field data on the concentration of respirable dust in air; and [e] corrections for the differences in bioavailability between particles that are swallowed vs. those retained in the lung (Table V).

TABLE V: Exposure Assumptions Used in Case I Calculations (Lifetime Average)

Route	CDC	Alternate
Oral	0.21 g/day ingested (lifetime ave.) 30% absorption	0.0028 g/day ingested (lifetime ave.) 10% absorption
Dermal	0.21 g/day of soil on the skin Dose is weighted by age over lifetime 1% absorption	0.5 mg soil/cm^2 skin 1400 cm^2 exposed surface 8 hr/day, 90 days/year 1% absorption
Inhalation	Total suspended particulates is 0.14 mg/m^3 15 m^3/day 100% in lungs 100% bioavailable	Total suspended particulates is 0.075 mg/m^3 15 m^3/day Of particles inhaled, assume 50% ingested and 50% in lungs Bioavailability weighted for differences in absorption for respirable and nonrespirable particles. Only 50% of the particles are from the contaminated site.

As shown in Table VI, using the alternate assumed values, the oral dose would not be expected to be greater than 2.8 fg/kg/day. If a 30% oral bioavailability were assumed, the average daily dose increases to 16.7 fg/kg/day. CDC's assumed values would suggest an oral uptake of about 606.5 fg/kg/day. The alternate method estimated dermal uptake of 2.7 fg/kg/day, in contrast to 20 fg/kg/day using CDC's assumed values. Lastly, CDC assumptions would suggest that 10 fg/kg/day might be inhaled versus 5.6 fg/kg/day. In summary, use of the alternative assumptions predicts that the most likely uptake of TCDD will, in this example, be 11.1 fg/kg/day. In contrast, the assumptions in

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

the CDC assessment led to an estimate for total TCDD uptake of 636.5 fg/kg/day (which CDC considered acceptable when recommending the 1 ppb guideline).

TABLE VI: Residential Site [a] (Case I)

	TCDD Uptake (fg/kg/day)	
Route	CDC	Alternate
Oral	606.5	2.8
Dermal	20.0	2.7
Inhalation	10.0	5.6
Total	636.5	11.1 (16.7) [b]

[a] Ten percent of land contaminated (10 ppb TCDD).
[b] Using a 30% oral bioavailability.

This analysis indicates that remediation would not be indicated at this site. Of course, in any isolated instances where children or adults might be exposed to TCDD soil concentration in excess of 500-1000 ppb, e.g., hot spots, clean-up of these would need to be considered. Concentrations as high as 1000 ppb in soil, assuming 1% dermal bioavailability, would not be expected to cause chloracne with repeated exposure, based on the work of Kligman who dermally applied TCDD to human volunteers (57,59).

CASE II (INDUSTRIAL SITE)

The CDC methodology can also be used to assess the potential hazards at an industrial site. In this example, assume that 20% of the site has been contaminated with 100 ppb TCDD. Because it is an industrial site, children have virtually no access. Is remediation necessary? The assumptions and factors used here are shown in Table VII.

TABLE VII: Exposure Assumptions Used in Case II Calculations (Lifetime Average)

Route	CDC	Alternate
Oral	Not relevant	Not relevant
Dermal	Exposure is almost same as oral exposure 15 m^3/day 100% in lungs	0.5 mg/soil 1400 cm^2 exposed skin 8 hr/day, 40 years exposure
Inhalation	0.14 mg/m^3 (dust) 15 m^3/day 100% in lungs 100% bioavailable	Total suspended particulates are 0.075 mg/m^3 (Springfield, Mo.) Of particles inhaled, assume 50% ingested and 50% in lungs 50% of respirable particles from contaminated soil Adjust bioavailability for ingestion and inhlation

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

Because children are not exposed, the oral route of exposure becomes insignificant. As shown in Table VIII, calculations based on the CDC assumed values indicate that an average lifetime dose of 590 fg/kg/day TCDD could potentially be absorbed by those who work at this site (lifetime average). This value is still less than 636 fg/kg/day, the daily dose which CDC considered acceptable (Table I). Further, use of the alternative assumptions indicates that the more likely daily uptake is 135 fg/kg/day. These calculations suggest, since the daily intake is less than 636 fg/kg/day, that 20% contamination of soil by 100 ppb TCDD at an industrial site poses no health hazard to workers or the community. Further, at many industrial sites it may be acceptable to permit soils contaminated with TCDD at levels greater than 100 ppb to remain in place, especially where soil erosion is not a problem and where the contamination is at the soil surface (where it is available for photodegradation if in the presence of solvents). Based on these factors, CDC noted that unless the levels of TCDD are greatly in excess of 100 ppb, paving should be an adequate remediation at many sites (1).

TABLE VIII: Industrial Site [a] (Case II)

	TCDD uptake (fg/kg/day)	
Route	CDC	Alternate
Oral	0	0
Dermal	390	78
Inhalation	200	57
Total	590	135

[a] 20% of land contaminated (100 ppb TCDD)

PROMOTER VERSUS INITIATOR

It is generally well accepted within the scientific community that tumor initiation involves a change to cellular DNA which is an irreversible and heritable event. Tumor promotion, on the other hand, involves reversible and nonheritable event(s). Promoters provide a favorable environment for tumor growth. When a promoter is taken away from a tumor, the environment becomes inhospitable, and tumor growth is adversely affected. In contrast, the alterations to DNA produced by initiators remain with that cell and are passed on to successive progeny cells during cell division.

The distinction between initiation and promotion as the mechanism of carcinogenesis is critical. Many in the scientific community would agree that one of the most critical aspects in the risk assessment of a carcinogenic substance is whether the substance acts through a genotoxic or non-genotoxic mechanism. Weisburger and Williams (58) point out the importance of this distinction: "The action of epigenetic agents of the promoter class is highly dose-dependent and reversible, and thus, a distinctly different risk analysis is required to take account of their quantitatively lesser hazard."

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

The discussion of the calculations in the previous sections is based on CDC's conclusion that an average daily uptake of about 636 fg/kg/day is a reasonable level at which to begin consideration of limiting exposure. In view of dioxin's lack of genotoxicity (60-64), however, this VSD seems excessive since it is based on the upper bound of risks estimated from models which assume linearity at low doses. Further, the arguments for linearity are frequently based on the chemical being genotoxic. Tumor promoters constitute a small percentage of the chemical carcinogens which have been studied or regulated. Regulatory agencies have much more experience in dealing with chemical initiators. Thus, the temptation is to regulate all carcinogens as initiators. However, if regulations are to be based on scientific fact and not on tradition, the cancer risk assessment for TCDD must reflect the fact TCDD is only a tumor promoter in animals. In fact, government bodies in Ontario, Germany and the Netherlands, many eminent scientists in Western Europe, and the U.S. Food and Drug Administration (FDA) regard TCDD as a promoter in animals and have estimated, accordingly, a lower potential risk to human health (see table 9).

By assuming TCDD is an initiator when the data attest to the contrary, EPA and CDC are not incorporating all the available data into their assessments. In EPA's "Proposed Guidelines for Carcinogen Risk Assessment" (65), they stipulate that models used in risk assessment should reflect data on mechanism of carcinogenesis:

> The choice of low-dose extrapolation models ***should be consistent with current understanding of the mechanisms of carcinogenesis*** . . . . When pharmacokinetics or metabolism data are available, or when other substantial evidence on the mechanistic aspects of the carcinogenesis process exists, a different low dose extrapolation model (than linear multi-stage) might be considered more appropriate on biological grounds. When a different model is chosen, the risk assessment should clearly discuss the nature and strength of the evidence that lead to the choice (emphasis provided).

For dioxin, there is evidence that its mechanism of action is receptor-mediated (66) and, to describe this mechanism, a low dose extrapolation model other than the linearized multi-stage is more appropriate. The linearized multi-stage model forces linearity at low doses - a trait which should not be forced for chemicals which act through a receptor mediated event. Further, the linearized models do not account for the reversible behavior of promoters or the possibility of a dose at which no increased risk would be expected.

TCDD LACKS INITIATOR ACTIVITY

The ability of TCDD to interact with genetic material has been examined by over 20 studies. Wassom et al. (60), Kociba (61-63), and Shu et al (64) have reviewed much of this data. Mutagenic

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

studies using the Salmonella/Ames bacteria test alone encompass 13 different strains with tests performed in 9 laboratories, including the National Toxicology Program. Of these independently conducted studies, all have been negative with the exception of two early studies by Hussain et al. (67) and Seiler (63). As has been discussed by Shu et al (64), there is good scientific reason to question the results of Hussain et al. and Seiler. These two early positive studies do not meet current criteria for demonstrating positive mutagenic response. For example, the studies did not demonstrate a dose response relationship; nor were the results reproducible by later studies. Moreover, in these studies, the TCDD used was greatly in excess of TCDD solubility in water, and the bacterial survival rates were extremely low.

In assessing the mechanism by which TCDD induces tumors in animals, regulatory agencies are expected to evaluate the largely negative mutagenesis findings on TCDD against the documented reliability which short term tests have for detecting mutagens and carcinogens. Several scientific panels have stated that it can be reasonably concluded that the negative mutagenic data on TCDD are real and not false negatives resulting from inadequate testing (69-70). The Salmonella/Ames test is extremely sensitive, and usually responds to micrograms, and in some cases even nanograms, of material. Given the sensitivity of the test, TCDD's lack of mutagenic activity is all the more convincing when it has been inactive in test after test. The importance of classifying a chemical as genotoxic in risk assessments has been quantitatively addressed in Squire (56), Park and Snee (71) and Longstreth and Hushon (55).

CANADIAN AND EUROPEAN VSD's

As early as 1981, Rodricks (72) suggested that in light of TCDD's lack of genotoxicity, the safety factor approach would be appropriate for setting an acceptable daily intake (ADI); using this approach, he calculated a 10 pg/kg/day figure. More recently, the Province of Ontario and several Western European countries, as well as European scientists, have estimated TCDD risk to humans that is significantly less than EPA's estimate. For example, the Ontario risk assessment uses a no observable effect level (NOEL) of 0.001 ug/kg/day (6,73) and a safety factor of 100 to obtain a maximum allowable daily intake of 1 X 10^{-11} g/kg/day (= 10 pg/kg/day) for humans (74). In contrast, EPA has used a value of 6.4 X 10^{-15} g/kg/day which suggests cancer risks that are 1670 fold higher. The United States Food and Drug Administration (FDA) has, on the other hand, accepted risks associated with the ingestion of up to 13 pg/kg/day. (Table IX).

The fundamental difference between the EPA and Canadian analyses is how each treats the data on TCDD's mechanism of action. Scientists from Canada and Western European countries regard TCDD as a tumor promoter in animals whereas EPA regards TCDD as a tumor initiator (75). The large body of data which indicate TCDD does not possess initiator activity but does possess promoter activity in animals has been discussed (60-64). The rather strong evidence that TCDD is a promoter rather than an

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

TABLE IX: TCDD Cancer Risk Estimates by Different Agencies

Agency	Acceptable risk	Model	Reference
Ontario	10,000 fg/kg/day	Safety factor (100)	Ontario, 1984 (74)
USEPA [a]	6.4 fg/kg/day	Linear multistage	EPA, 1985b (51)
CDC [b]	28-1428 fg/kg/day	Linear multistage	Kimbrough *et al.*, 1984 (1)
	632.6 fg/kg/day	(Best estimate)	
FDA	13,000 fg/kg/day	Safety factor (77)	Cordle, 1981 (107)
		Linear multistage (10^{-5} risk, mle)	

[a] Acceptable defined as 10^{-6} risk (upper bound).
[b] Based on mouse and rat bioassay (10^{-6} risk).

initiator, and the likelihood that current dose-response models may overstate the actual risks, were acknowledged by Kimbrough et al (1).

Extrapolating To Very Low Doses

As discussed by Sielken (76), the method used to extrapolate the dose-response curve can be an important part of any risk assessment. The scientist responsible for selecting the most appropriate method for extrapolating the dose-response curve faces a multitude of uncertainties which require several choices, assumptions and judgments during that selection process. These decisions generally will have serious impact on the results and have a marked effect on the risk management decision. In short, the choice of model, tumor type, species, bounding procedures, etc. can radically impact the results of the extrapolation process. These decisions are often driven more by policy than by the available scientific information. Before the results of the extrapolation process are allowed to influence risk management decision, a thorough analysis of the quantitative impact of the particular choices made in an assessment should be presented and the scientific merit of these particular choices should be carefully explained (54).

The interpretation of the dose-response curve for dioxin, like the dose-response relationship for other chemicals, requires careful thought since numerous subtleties can dramatically affect the results. For example, false impressions can arise from fitting quantal response models to very high administered doses. In the Kociba et al study (6), the highest experimental dose level (0.1 ug/kg/day) is one hundred times larger than the lowest non-zero experimental dose level (0.001 ug/kg body wt/day). Even the second highest experimental dose level (0.01 ug/kg body wt/day) is ten times the lowest non-zero experimental dose level. As noted by Sielken (76), the trade-offs inherent in curve fitting may lead to questionable fits in the low-dose region of primary concern.

The fitted models in Figure 3 reflect trade-offs both in the sense that the fitted models are shaped differently from the experimental data and in the sense that the fitted model response rates, relative to the observed rates, are too large at the lowest non-zero experimental dose level and too small at the intermediate non-zero level. Furthermore, the need to accommodate the highest experimental dose forces the fitted

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

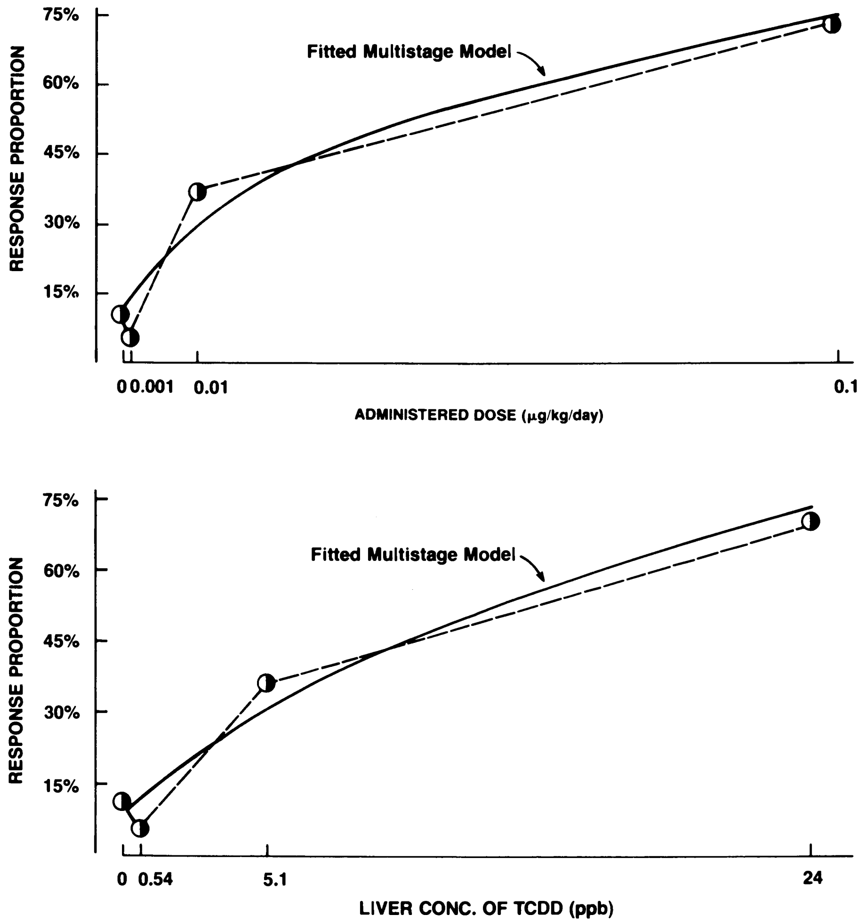


Figure 3: Regardless of whether the dose is expressed as ADMINISTERED DOSE or LIVER CONCENTRATION, when the multistage model is fit to the experimental data on hepatocellular neoplastic nodule or carcinoma in female rats in the Kociba et al (1978) study, the fitted models do NOT reflect the observed behavior at the lower experimental doses (From ref. 76). Reproduced with permission from ref. 108. Copyright 1986, Academic Press.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

models to be very non-responsive to whatever is observed at the lowest non-zero dose level. For example, as illustrated in Figure 4, the presence or absence of the experimental data at the lowest experimental dose has very little effect on the overall shape of the fitted models and makes only a relatively small effect (factor of 2) on the fitted model values for the VSD.

For several years, it has been acknowledged that whenever possible bioassay data should be corrected for early death. In both CDC's and EPA's analysis of Kociba et al (6), the individual animal pathology results were not available. Recently, these data were made available and the analysis showed some interesting results (76-77). The nonparametric Kaplan-Meier estimates of the probability of a female rat developing a hepatocellular neoplastic nodule or carcinoma by the end of 25 months, the duration of the Kociba study, are shown in Figure 5. These estimates are computed separately for each dose level and take into account the observation times of each rat. The values of these estimates for the two highest dose levels are 0.81 at 0.01 ug/kg/day and 1.00 at 0.1 ug/kg/day. Thus, by the end of the experimental period the number of rats that could develop a hepatocellular response is completely saturated at 0.1 ug/kg/day and nearly saturated at 0.01 ug/kg/day (76). It is impossible for the multi-stage model to portray both this type of saturation phenomenon and the observed nonlinearity at the lowest experimental dose levels (0, 0.001, and 0.01 ug/kg/day). The conflict caused by the model's inability to reflect both behaviors is dealt with in the fitting process by essentially ignoring the lower dose behavior and focusing on depicting the relative flatness at higher doses.

As discussed by Sielken (76), the flattening out or leveling off of the observed dose-response relationship at high doses causes the fitted models to modify dramatically their lower dose shape in order to have a relatively flat higher dose shape. This change in the shape of the fitted models at the lower doses is illustrated in Figure 6 where the fitted multi-stage model on the administered dose scale is relatively linear if 0.1 ug/kg body wt/day is included in the fitting process and is upward curving in the lower dose region if 0.1 ug/kg body wt/day is excluded. These two fits have very different shapes in the lower dose region, particularly between 0 and 0.001 ug/kg/day. Consequently, the use of the very high dose data here leads to fitted models with an unsupported false impression of linearity in the lower dose region. This pitfall is lessened by fitting the models to the observed proportions excluding the highest dose level.

When the very highest experimental dose level is not allowed to dominate the shape of the fitted multi-stage model for hepatocellular neoplastic nodule or carcinoma in the Kociba study, the nonlinearity in the fit of the multi-stage model to the three lower experimental dose levels is much more consistent with the observed nonlinearity in the fitted multi-stage model for the same carcinogenic response in the NTP study (Figure 7). Contradictory shapes arise (Figure 8) if the data at the high dose in the Kociba study are allowed to dominate the fit.

In short, the availability of the time-to-early-death data for the animals in the Kociba study, recognition of the inability

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

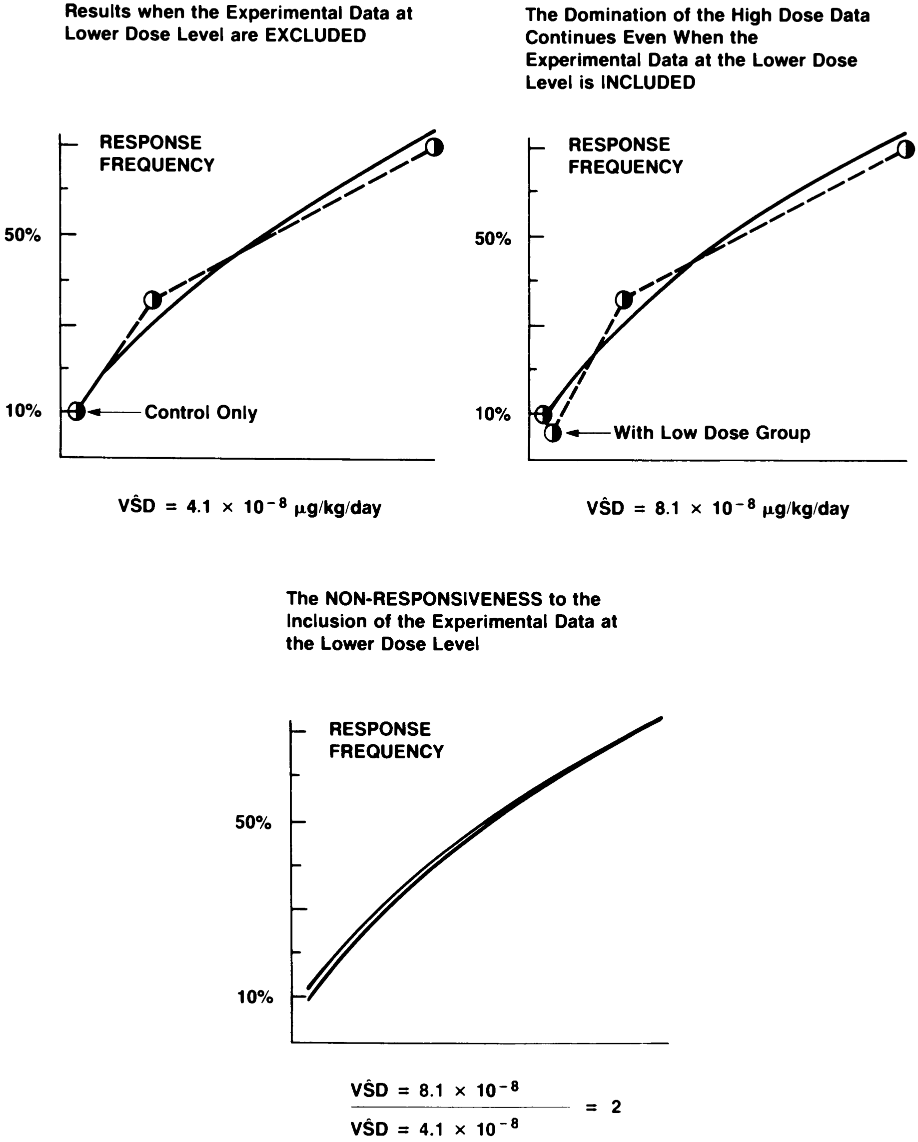


Figure 4: Both the fit of the multistage model to the Kociba et al (1978) experimental data on hepatocellular neoplastic nodule or carcinoma in female rats and the corresponding estimate of the virtually safe dose (VSD) are dominated by the higher experimental dose levels and are ESSENTIALLY UNAFFECTED by the results obtained at lower doses (From ref. 76). Reproduced with permission from Paustenbach et al. Copyright 1986, Academic Press.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

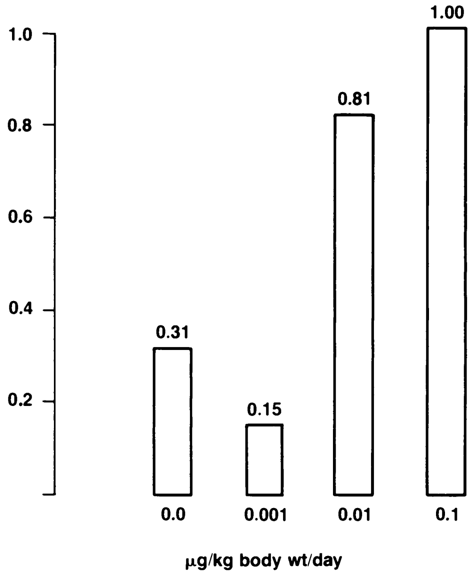


Figure 5: The Kaplan-Meier estimates of the probability of a female rat developing a hepatocellular neoplastic nodule or carcinoma in the Kociba et al (1978) study suggest that the dose-response relationship resembles one with a saturation-like phenomenon occurring at the highest experimental dose level (From ref. 76). Reproduced with permission from Paustenbach et al. Copyright 1986, Academic Press.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

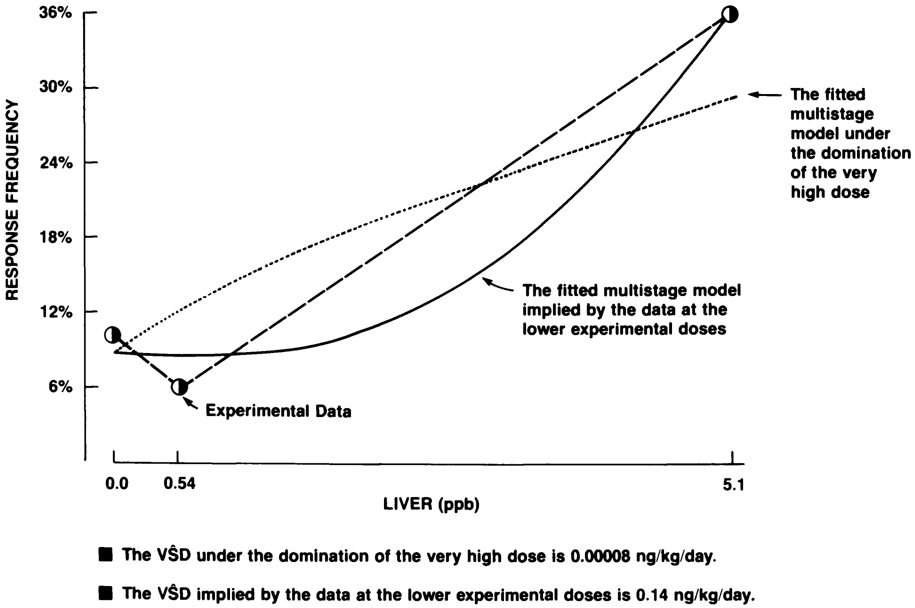


Figure 6: The fit of the multistage model and the estimated virtually safe dose (VSD) for the Kociba et al (1978) data on hepatocellular neoplastic nodule or carcinoma in female rats are substantially different (1750 fold) if the domination of the very high dose is removed (From ref. 76). Reproduced with permission from Paustenbach et al. Copyright 1986, Academic Press.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

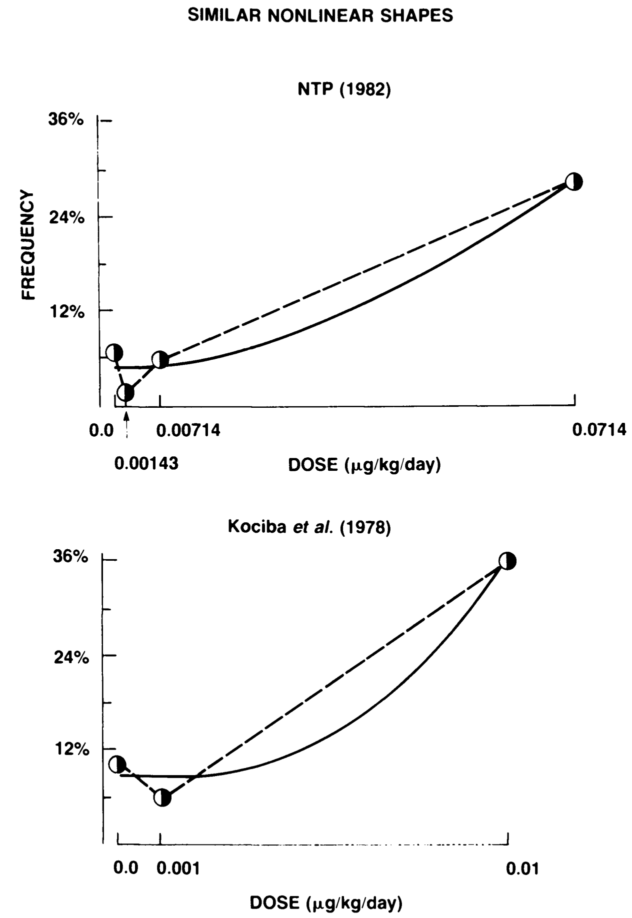


Figure 7: The shape of the fitted multistage model for the experimental data on hepatocellular neoplastic nodule or carcinoma in female rats in the NTP (1982) study SUPPORTS the NONLINEARITY in the shape of the fitted multistage model for the experimental data on hepatocellular neoplastic nodule or carcinoma in female rats at the lower doses in the Kociba et al (1978) study (From ref. 76). Reproduced with permission from Paustenbach et al. Copyright 1986, Academic Press.

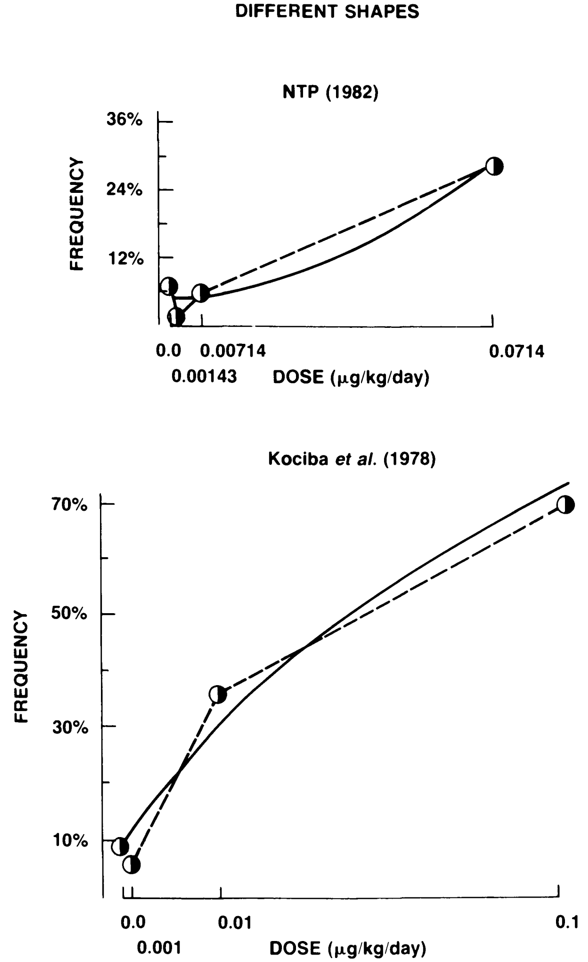


Figure 8: The shape of the fitted multistage model for the experimental data on hepatocellular neoplastic nodule or carcinoma in female rats in the NTP (1982) study does NOT SUPPORT the LINEARITY in the shape of the fitted multistage model for the experimental data on hepatocellular neoplastic nodule or carcinoma in female rats in the Kociba et al (1978) study if the data at the very high dose is allowed to dominate the fit (From ref. 76). Reproduced with permission from Paustenbach et al. Copyright 1986, Academic Press.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

of the multi-stage model to respond to the data and the domination of the highest dose in the model provides grounds for re-evaluating the prior mathematical analysis of the cancer bioassay data. Specifically, the corresponding estimated virtually safe dose (VSD) for an increase of 0.000001 (one in a million) in the probability of hepatocellular neoplastic nodule or carcinoma in a female rat is 140 pg/kg/day (140,000 fg/kg/day) in the diet using the Kociba data at 0.001 and 0.01 ug/kg/day and is 130 pg/kg/day in the diet using the NTP data. The corresponding estimated VSD's for male rats are higher, as are those for hepatocellular carcinoma alone.

UPTAKE BY CROPS

The likelihood that crops will take-up (absorb) appreciable quantities of TCDD from contaminated soil is negligible (51). Oats and soybean plants grown to maturity in soil contaminated with 0.06 ppm 2,3,7,8-TCDD showed less than 1 ppb of 2,3,7,8-TCDD in the seeds (78). Cocucci et al. (79) measured the level of contamination in kitchen garden plants (carrot, potato, onion and narcissus) grown in soil from the contaminated Seveso area containing 1000-4000 mg/m^2 of 2,3,7,8-TCDD. Although dioxin was found to be 3-5 times higher in foliage than in fruits, these results have been questioned because others have not made similar observations. Wipf et al. (80) failed to detect any measurable 2,3,7,8-TCDD in the flesh of fruits and vegetables collected from the contaminated area in Seveso during 1977-1979, although the soil 2,3,7,8-TCDD concentration was 10 ppb. These authors concluded that 2,3,7,8-TCDD does not appear to be translocated from soil to the plants. A similar conclusion was reached by Pocchiari et al. (81) from their uptake experiments with plants. As noted by EPA, it can be concluded from these studies that 2,3,7,8-TCDD will not accumulate in plants grown in contaminated soils (51).

EXPOSURE OF DOMESTIC ANIMALS

The uptake and translocation of TCDD in plants used for animal feed is not an important route of animal exposure (82). Although Fries has noted that residues of TCDD are occasionally reported in the aerial parts of plants grown in contaminated soil (82), the more usual finding has been that there is little or no TCDD in the aerial parts and seeds of crops grown in contaminated soil (83,84). Results from work on TCDD are substantially in agreement with more extensive work on other halogenated hydrocarbons (80). The latter studies generally indicate that contamination of the aerial parts of the plant is mainly from surface contamination due to dust or redeposition of volatilized material from soil. Field experience with PBB indicates that surface contamination from dust gathered during harvest of forage crops makes a negligible contribution to residues in feed as harvested. This concentration was less that 1% of that present in soil (85).

The ingestion of soil by grazing animals is apparently the

main route by which livestock take up many environmental contaminants which are contained on particles (e.g., granular pesticides and contaminated dust). This is in contrast with the belief that the primary hazard is due to the ingestion of contaminated foods or crops. Soil ingestion by cattle and sheep has been studied under a variety of conditions (86-92). It has been shown that soil ingestion is inversely related to the availability of forage (82). The amount is as low as 1-2% of dry matter intake in seasons of lush plant growth and it rises as high as 18% in periods when forage is sparse. Under New Zealand conditions, where animals can graze 365 days a year, average soil intake was about 6% of the estimated dry matter intake for cattle and 4.5% of the dry matter intake for sheep (93). The amount of soil ingested is reduced more than 50% when animals are offered harvested feed as a supplement to pasture (88-89).

Farm animals also ingest soil when they are confined to unpaved holding areas (86,91). Under typical U.S. farm conditions, lactating cows may consume as much as 1% of their dry matter intake as soil whereas nonlactating cattle, who have greater exposure and are less intensely fed, may consume up to 4% of their dry matter intake from this source. Pigs because of their habit of rooting will tend to have higher values, which can be as great as 8% of dry matter intake (86). Since pigs never subsist on pasture alone, this value may be considered an upper limit for the amount of soil ingested by pigs.

The relationship between the concentration of TCDD in soil and the ultimate food product is determined largely by the amount of soil ingested by animals and this is determined in large part by the animal management system. Farmers who primarily eat their own livestock will be the population who will be exposed to the highest levels of TCDD and many of them will have other sources of meat. When estimating the amount of TCDD which could be detected in meat or milk, several basic factors must be considered: [a] amounts of soil ingested by the animal (yearly averages), [b] the fat:diet ratio is usually 5:1 for milk and body fat, [c] the amount of soil ingested should be averaged over a year when animal access is less than a full year, [d] the half-life of TCDD in growing cattle is 115 days, [e] the degradation of TCDD-contaminated soil at the soil surface, [f] rate of metabolism by the animal, and [g] soil bioavailability in the animal. (94-99).

Fortunately, the practical significance of soil ingestion as a potential route of animal exposure is greatly mitigated under the U.S. agricultural conditions. (82) Lactating dairy cows are rarely pastured and some form of supplemental feeding is always employed. Consequently, it is unlikely that soil ingestion would ever exceed 1 or 2% of dry matter intake in actual farm situations. While Fries has noted that cattle raised for beef might often be on pastures with little other feed, it is the general practice to fatten these animals in feed lots before slaughter. This period of time may be as long as 150 days and animals can gain as much as 60 to 70% in body weight. (87) TCDD concentrations would also be reduced by dilution in the expanding body fat pool. The role of metabolism is very important for

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

animals which might be eaten. Recent data from Texas A&M (100) indicate that the cow readily and thoroughly metabolizes TCDD - a trait not possessed by most other species.

Most hogs destined for slaughter are confined and would never be exposed to contaminated soil. Thus, as noted by Fries (82) only cull breeding cattle and pigs might be expected to go directly from soil to slaughter and these are not the animals ordinarily used for home consumption by farmers who raise them. In short, for most domestic animals in the United States, few will ever be exposed to contaminated soil without having been provided with ample feed. In general, only when they graze upon soil where the dioxin level exceeds 1-20 ppb might it pose a hazard to those persons who primarily eat their own meat products. For nearly all others, due to dilution with other meats from numerous sources, as well as the other factors which we have discussed, the risk will be de minimus. Due to the many factors that need to be considered, the setting of an acceptable soil level should be done on a site specific basis.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

DISCUSSION

The selection of the most appropriate assumptions is critical in any environmental risk assessment. Often, as few as one, two or three assumptions or factors can dominate the results of the assessment. Clearly, in the health assessment of dioxin in soil, the most critical parameters are the quantity of soil ingested by children and adults, the results of the mathematical modeling of the bioassay data, consideration for dioxin's lack of genotoxicity, and dioxin's lower bioavailability in a soil matrix. Other assumptions used in the assessment are also important but generally they do not alter the results by 2-3 orders of magnitude as do those which have been discussed.

The application of more sophisticated approaches to quantitative risk assessment for cancer and other toxic effects such as physiological-pharmacokinetics should markedly reduce the uncertainties in the health hazard assessment process for dioxin (102) and these should be incorporated when available. In addition, regulatory decisions regarding the appropriateness of adopting risk criteria such as 1 in 1,000,000 must be carefully considered with respect to dioxin contaminated soil in light of the relatively small number of persons exposed to significant levels. For example, most environmental and occupational regulations that have been promulgated thus far, tend to accept cancer risks in the region of 1 in 1,000 to 1 in 100,000 since it is recognized that these risk assessment models are believed to overestimate the true risks (based on human data) or because the exposed population is so small that cost/benefit criteria indicate that lower risks are financially impractical (102,103).

EPA has recently found the maximum individual risks and total population risks from a number of radionuclide and benzene sources to be too low to be properly described as "significant". Specifically, benzene emissions from maleic anhydride process vents created maximum individual risks of 7.6 in 100,000 and an aggregrate yearly cancer incidence of twenty-nine thousandths of

a case (104). Radionuclides from the Dept. of Energy facilities would expose a person who accrued lifetime exposure to a plant's most concentrated emissions to a risk of 1 to 8 in 10,000, while, in the aggregate, only eight-hundredths of a cancer would be predicted to occur yearly (105). EPA found these risks to be insignificant and eventually withdrew the proposed regulations (106).

CDC, in its written assessment and in all of its public discussions on the topic, has urged that regulatory agencies understand the underpinnings of the methodology and its assumptions before clean up measures are decided upon. Use of the more justifiable assumptions, which we have discussed, should assist regulatory agencies in developing a more rational and scientifically supportable approach to the remediation of TCDD-contaminated sites (108).

In summary, the CDC assessment proposed a site-specific recommendation, not a universal standard for clean-up. CDC's analysis assumed a uniform contamination of the entire soil surface in residential neighborhoods, a situation not known to exist in Missouri or elsewhere in the United States. Further, the CDC 1 ppb guideline and risk assessment methodology were intended for application only to residential sites--not to industrial sites or other areas where the exposure to children would not occur. The present assessment indicates that, depending on the site characteristics and the use patterns, a soil concentration of TCDD considerably in excess of 1 ppb is acceptable for residential areas. Soil concentrations greater than 100 ppb in non-residential areas should amply protect the environment and public health.

Literature Cited

1. Kimbrough, R.; Falk, H.; Stehr, P.; Fries, G. J Toxicol Env Health 1984, 14, 47-93.
2. Schaum, J. "Risk Analysis of TCDD Contaminated Soil"; Office of Health and Environmental Assessment, U.S. Environmental Protection Agency; Washington, D.C., 1983.
3. Houk, V. Chemosphere 1986, 15, 1875-1881.
4. St. Louis Globe-Democrat. "Castlewood dioxin site clean-up to cost more than $12.7 million." July 20, 1985.
5. Kay, M. Abstract 32. 196th meeting of the American Chemical Society. (Symposium on Solving Hazardous Waste Problems.) 1986.
6. Kociba, R.J.; Keyes, D.G.; Beyer, J.E.; Carreon, R.M.; Wade, C.E.; Dittehber, D.A.; Daknins, R.P.; Frauson, L.E.; Park, C.N.; Barnard, S.D.; Hummel, R.A.; Humiston, C.G. Toxicol Appl Pharmacol, 1978, 46, 279-303.
7. "National Toxicology Program, Technical Report #209", Research Triangle Park, NC, 1980.
8. Portier, C.J.; Hoel, D.G.; Van Ryzin, J. In "Public Health Risks of the Dioxins", W.W. Lowrance, ed. William Kaufmann, publishers, Los Altos, Calif, 1984: Chapter 7.
9. Walter, S.D.; Yankel, A.J.; von Lindern, I.H. Arch Env Health 1980, 35, 53-58.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

10. Cooper, M. "Pica"; Charles C. Thomas Publishers. Springfield, IL, 1957; p. 65-66.
11. Charney, E.; Sayre, J.; M. Coulter; Pediatrics 1980, 65, 226-231.
12. Sayre, J.W.; Charney, E.; Vostal, J.; Pless, B. Am J Dis Child 1974, 127, 167-170.
13. Duggan, M.J; Williams, S. In "The Science of the Total Environment", 1977, 7, 91-97.
14. Lepow, M.L.; Bruckman, L.; Robino, R.A.; Markowitz, S.; Gillette, M.; Kapish, J. Environ Health Perspectives 1974, 6, 99-101.
15. Lepow, M.L.; Bruckman, L.; Gillette, M.; Markowitz, S.; Robino, R.; Kapish, J. Env Res 1975, 10, 415-426.
16. Barltrop, D. "Proceedings Inter Symp Env Health Aspects of Lead". Published by Commission of European Communities, Center for Information and Documentation, Luxembourg, 1973.
17. "Lead in the human environment". National Academy Press, 2101 Constitution Avenue NW, Washington, DC. 20418, 1980.
18. Day, J.P.; Hart, M.; M.S. Robinson. Nature 1975, 253, 343-345.
19. Bryce-Smith, D. Phys. Bull., 1974, 25: 178.
20. "Air Quality Criteria for Lead". Vol. II. U.S. Environmental Protection Agency, 1984, EPA-600/8-83-028A.
21. Hawley, J. Risk Analysis 1985, 5, 289-302.
22. Bellinger, D.; Leviton, A.; Rabinowitz, M.; Needleman, H.; C. Waternaux. Pediatrics 1986 (Submitted).
23. Binder, S.; Sokal, D.; Maughan, D. Arch. Env. Health 1987 (in press).
24. Clement Assoc., Inc. "Endangerment assessment for the Smuggler Mountain site, Pitkin County, Colorado". Prepared for the U.S. EPA (Region VIII), under Contract No. 68-01-6939, 1986.
25. O. Clausing; A.B. Brunekreef; van Wijnen, J.H.. Inter. Arch. Env. Occ. Health 1987 (in press.)
26. Russell, R.S. In "Radioactivity and human diet". (R.S. Russell, ed.) Pergamon Press Inc.: Long Island City, New York, 1966; Chapter 5.
27. "Airborne lead in perspective". National Academy of Science: Washington, D.C., 1972.
28. Romney, E.M.; Lindberg, N.G.; Hawthorne, H.A.; Bystrom, B.B.; Larson, K.H. Ann. Rev. Plant Physiol. 1963, 14, 271-279.
29. Martin, W.E. Radiation Bot. 1964, 4, 275-281.
30. Superfund Health Assessment Manual (draft). Submitted to The Office of Emergency and Remedial Response, U.S. Environmental Protection Agency. Produced by ICF Incorporated under EPA contract 68-01-6872, 1985.
31. Wolfe, H.R.; J.F. Armstrong; W.F. Durham. Mosquito News, 1974, 34, 263-267.
32. Trijonis, J.; Eldon, J.; Gins, J.; Berglund, G. Analysis of the St. Louis RAMS Ambient Particulate Data. Produced by Technology Service Corporation under EPA contract 68-02-2931 for the Office of Air, Noise, and Radiation of the U.S. Environmental Protection Agency. EPA report 450/4-80-006a, 1980.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

33. Environmental Protection Agency (EPA) of Region VII. Correspondence from John Helvig, 1985.
34. Environmental Protection Agency (EPA). Air quality criteria for particulate matter and sulfur oxides. Volume 2. U.S. Environmental Protection Agency, Office of Environmental Criteria and Assessment. Research Triangle Park, N.C. EPA-600-8-82-029bF. 1982. P. 5-106 to 5-112.
35. Poiger, H.; C. Schlatter. Food Cosmetic Toxicol., 1980, 18, 477-481.
36. Bartek, M.J.; LaBudde, J.A. Percutaneous absorption, in vitro. In "Animal Models in Dermatology". H. Maibach, ed. Churchill Livingston: New York, 1975; pp 103-120.
37. Wester, R.C.; Noonan, P.K. Int. J. Pharm., 1980, 7, 99-110.
38. Roels, H.; Buchet, J.P.; Lauwerys, R.R. Environ. 1980, 22, 81-94.
39. Brunekreef, B.; Noy, D.; Biersteker, K.; Boleij, J. J. Air Pol Control Assn 1983, 33, 872-876.
40. Snyder, W.S. "Report of the Task Group on Reference Man". International Commission of Radiological Protection No. 23. Pergammon Press: NY, 1975.
41. Philippi, M.; Krasnobagew, V.; Zeyer, J.; Huetter, R. In "Microbial Cultures and Soil Under Laboratory Conditions". Fems Symp 1981, 12, 2210-2233.
42. Huetter, R.; Philippi, M. Pergammon Ser Environ Sci 1982, 5, 87-93.
43. McConnell, E.; Lucier, G.; Rumbaugh, R.; Albro, P.; Harvan, D.; Hass, J.; Harris, M. Science 1984, 223, 1077-1079.
44. Lucier, G.W.; Rumbaugh, R.C.; McCoy, Z.; Hass, R.; Harvan, D.; Albro, P. Fund. Appl. Tox. 1968, 6, 364-371.
45. Umbreit, T.H.; Hesse, E.J.; Patel, D.; Gallo, M.A. Presented at the annual meeting of the Society of Toxicology, San Diego, CA (Abstract 249), 1985.
46. Umbreit, T.H.; Hesse, E.J.; Gallo, M.A. Science 1986, 232, 497-499.
47. Umbreit, T.H.; Hesse, E.J.; Gallo, M.A. Presented at the annual meeting of the Society of Toxicology, New Orleans, LA (Abstract 1273), 1986.
48. Environmental Reporter. Bioavailability said to vary. June 28, 1985, p. 328.
49. Chemical Regulation Reporter. "Some assumptions used in risk assessment for Missouri areas were incorrect, Houk says." (March 18th), 1986, p. 63.
50. Bonaccorsi, A.; di Domencio, A.; Fanelli, R.; Merli, F.; Motta, R.; Vanzati, R.; Zapponi, G.A. Arch Toxicol. Suppl. 1984, 7, 431-436.
51. "Health Assessment Document for Polychlorinated Dibenzo-p-dioxins. (Final Report)". U.S. Environmental Protection Agency, Office of Env. Assessment, Cincinnati, Ohio. EPA-600/8-84-014F, 1985b, P. 5-8 and 5-10.
52. Yanders, A.F. Presented at the Maine Department of Environmental Protection Dioxin Workshop. Augusta, Maine (Feb. 4th), 1986.
53. Di domenici, A.; Silano, V.; Viviano, G.; Zapponi, G. Ecotoxicol Environ Safety, 1980, 4, 298-320.
54. "Risk Assessment in the Federal Government: Managing the

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

Process". National Academy of Sciences, National Academy Press: Washington, DC, 1983.
55. Longstreth, J.D. and Hushon, J.M. In: Tucker, R.E., Young, A.L., Gray, A.P. eds. "Human and Environmental Risks of Chlorinated Dioxins and Related Compounds", Plenum Press: NY, 1983, p. 639-664.
56. Squire, R.A. Science 1981, 214, 877-880.
57. Tschirley, F.H. Scientific American 1985, 254, 29-35.
58. Weisburger, J.H.; Williams, G.M. Fd Cosmet Toxicol 1981, 19, 561-566.
59. Young, A. Bull Environ Contam Toxicol 1984, 33, 702-709.
60. Wassom, J.S. Mutat. Res. 1977, 47, 141-160.
61. Kociba, R.J. In: "Banbury report 18: Biological mechanisms of dioxin action". A. Poland, R. Kimbrough, ed. Cold Springs Harbor, NY, 1984, P. 73-84.
62. Kociba, R.J. Abstract #78. 5th Inter. Symposium on Dioxin and Related Compounds(Bayreuth, Germany), 1985.
63. Kociba, R.J. Chemosphere 1986, in press.
64. Shu, H.P.; Paustenbach, D.J.; Murray, F.J. Regul Toxicol Pharm. 1987, (in press).
65. EPA Proposed Guidelines for Carcinogen Risk Assessment. Federal Register 51 CFR 29844, Sep. 24, 1986, p. 33992-34003.
66. Poland, A. 1st Annual Burroughs Welcome Lecture, Meeting of the Society of Toxicology, New Orleans, LA; March 6, 1986.
67. Hussain S.; Ehrenberg, L.; Lofroth, G.; Gejvall, T. Ambio 1975, 1, 32-33.
68. Seiler, J.P. Experientia 1973, 29, 622-623.
69. California Scientific Review Panel Discussions (1985). Health effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin and related compounds: Response to public comments. Department of Health Services, Berkeley, California, 1985.
70. EPA Science Advisory Board, Environmental Health Committee Transcripts. Nov. 29, 1984.
71. Park, C.; Snee, R. Fund Appl Toxicol 1983, 3, 320-333.
72. Rodricks, J.V. In "Human and Environmental Risks of Chlorinated Dioxins and Related Compounds". Tucker, R.G., Young, A.L. and Gray, A.P., Eds. Plenum Press: New York, 1983.
73. Murray, F.J.; Smith, F.A.; Nitschke, K.D.; Humiston, C.G.; Kociba, R.J.; Schwetz, B.A. Toxicol Appl Pharmacol 1978, 50, 241-251.
74. "Scientific Criteria Document for Standard Development No. 4-84: Polychlorinated Dibenzo-p-dioxins (PCDD's) and Polychbrinated Dibenzofurans (PCDF's)". Ontario Ministry of the Environment. September, 1985.
75. Whittemore, A.S.; S.C. George; Silver, A. Fund Appl Toxicol 1986, 7, 183-190.
76. Sielken, R.L. Food And Chemical Toxicology 1987, (in press).
77. Sielken, R.L.; Carlborg, F.W.; Paustenbach, D.J.; Shu, H.P.; Murray, F.J. (Abstract 1133). Presented at the 25th Annual Meeting of the Society of Toxicology. New Orleans, LA, 1986.
78. Isensee, A.R.; G.E. Jones. J. Agric. Food Chem. 1971, 19, 1210-1214.
79. Cocucci, S.; F. Di Gerolamo; A. Verderio, et al. Experientia. 1979, 35(4), 482-484.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

80. Wipf, H. K.; Homberger, E.; Neimer, N.; Ranalder, U.B.; Vetter, W.; Vuilleumeir, J.P. In: "Chlorinated Dioxins and Related Compounds: Impact on the Environment", O. Hutzinger, et al., Ed. Pergamon Press: NY, 1982, p. 115-126.
81. Pocchiari, F.; A. DiDomenico; V. Silano; G. Zapponi. In "Accidental Exposure to Dioxins: Human Health Aspects", F. Coulston and F. Pocchiari, Ed. Academic Press: NY, 1983, p. 5-35.
82. Fries, G. F. (Abstract GL-02), 6th Inter. Symposium on the Chlorinated Dioxins and Related Compounds. (Fukuoka, Japan), 1986.
83. Jensen, D.J.; Getzendaner, M.E.; Hummel, R.A.; Turley, J. J. Agric. Food Chem. 1983, 31, 118-122.
84. Sundstrom, G.; Jensen, S.; Jansson, B.; K. Erne. Arch. Environ. Contam. Toxicol. 1979, 8(4), 441-448.
85. Fries, G.F.; Jacobs, L.W. Mich. State Univ. Agric. Exp. Sta. Res. Report. 1986, 477.
86. Fries, G.F.; Marrow, G.S.; Snow, P.A. Environ. Toxicol. Chem. 1982, 1,201-204.
87. Healy, W.B. N.Z. J. Agric. Res. 1968, 11,487-499.
88. Healy, W.B.; Cutress T.W.; Michie C. N.Z. J. Agric. Res. 1967, 10, 201-209.
89. Healy, W.B.; Drew, K.R. N.Z. J. Agric. Res. 1970, 13, 940-944.
90. Healy, W.B.; Ludwig, T.G. N.Z. J. Agric. Res. 1965, 8, 737-752.
91. Fries, G.F.; Marrow, G.S.; Snow, P.A. J. Dairy Sci. 1982, 65, 611-618.
92. Mayland, H.F.; Florence, A.R.; Rosenau, R.C.; Lazar, V.A.; Turner, H.A. J. Range Manage 1975, 28, 448-452.
93. Fries, G.F. J. Toxicol. Environ. Health 1985, 16, 565-579.
94. Jensen D.J.; Getzendaner, M.E.; Hummel R.A.; Turley, J. J. Agric. Food Chem. 1983, 31, 118-122.
95. Jensen D.J.; Hummel, R.A.; Mahle, N.H.; Kocher C.W.; Higgins, H.S. J. Agric. Food Chem. 1981, 29, 265-268.
96. Jensen, D.J.; Hummel, R.A. Bull. Environ. Contam. Toxicol. 1982, 29, 440-446.
97. Kocher, C.W.; Mahle, N.H.; Hummel, R.A.; Shadoff, L.A.; Getzendaner, M.E. Bull. Environ. Contam. Toxicol. 1978, 19, 229-236.
98. Jensen D.J.; Hummel, R.A.; Mahle, N.H.; Kocher C.W.; Higgins, H.S. J. Agric. Food Chem. 1981, 29(2), 265-268.
99. Fries, G.F. J. Environ. Qual., 1982, 11, 14-20.
100. Safe, S.; Ivie, D. (Abstract BP-20). 6th Inter. Dioxin Symposium (Fukuoka, Japan), 1986.
101. Andersen, M.E.; Gargas, M.; Reitz, R.A. Toxicol. Appl. Pharm 1987, in press.
102. Ruckelshaus, W.D. Vital Speeches of the Day. City News Publishing Co.: Southold, NY, April 1st, 1984.
103. Bartman, T.R. In "Quantitative Risk Assessment in regulation". L.B. Lave, Ed. The Brookings Institution Press: Washington, D.C., 1982.
104. "National Emission Standards for Hazardous Air Pollutants; Benzene Emissions from Maleic Anhvdride Plants,

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

Ethylbenzene/Styrene Plants, and Benzene Storage Vessels; Proposed Withdrawal of Proposed Standards", 49 Fed. Reg. 8386, 8388 (March 6, 1984) ("Benzene NESHAPS Withdrawal Notice")

105. "National Emission Standards for Hazardous Air Pollutants; Regulation of Radionuclides; Withdrawal of Proposed Standards", 49 Fed Register 43906, 43910 (Oct. 31) ("Radionuclides NESHAPS Withdrawl Notice").
106. Wreen, G. Asbestos ban and phaseout proposal. Testimony before OSHA (June 29th). Transcript available from Environ Corp, 1000 Potomac, The Flour Mill, Washington, D.C., 1986.
107. Cordle, F. Regul Toxicol. Pharm. 1981, 1,379-387.
108. Paustenbach, D.J.; Shu, H.P.; Murray, F.J. Regul Toxicol Pharm 1986, 6, 284-307.

RECEIVED February 4, 1987

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch014

Chapter 15

Solving Dioxin Contamination Problems in Missouri

Morris Kay and Ralph Hazel

U.S. Environmental Protection Agency, Region VII, 726 Minnesota Avenue, Kansas City, KS 66101

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch015

This paper discusses measures taken by U. S. Environmental Protection Agency (EPA) Region VII, to deal with the problem of dioxin contamination in Missouri. Investigations which were carried out to identify the sites of contamination are described, along with temporary measures which were taken to protect public health and the environment from the dioxin contamination. The Region's participation in an accelerated research effort to find solutions to the problem is detailed, particularly a project whereby the EPA mobile incinerator was brought to a farm site in Southwest Missouri to demonstrate its effectiveness in destroying dioxin. Finally, the paper describes cleanup activities which have been completed or are well underway as the Agency moves rapidly toward a comprehensive, final solution to the Missouri dioxin problem.

The Environmental Protection Agency (EPA) working with the state and other federal agencies has moved rapidly to clean up dioxin contamination in Missouri. Since the buy out of Times Beach in 1983, EPA has developed safe clean-up technology to remove dioxin-laden soil and destroy the dioxin by incineration. In addition, EPA has successfully removed dioxin materials from residences and streets and rehabilitated previously contaminated property.

More than 40 sites have been identified in the state of Missouri where soil is contaminated with 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) at levels above 1.0 parts per billion. Many of the sites resulted from dioxin-containing wastes being mixed with waste oil and sprayed as a dust suppressant. Other sites such as the Minker residential area were contaminated by using soil as fill dirt which had been sprayed for dust control and excavated and moved from a horse arena.

Knowledge of the dioxin spraying activities did not fully become known to Region VII EPA until 1982 when a full investigation was

This chapter not subject to U.S. copyright.
Published 1987, American Chemical Society

started. Since that time two hundred fifty-two (252) potential sites have been investigated in the State. In the course of these investigations, 13,593 soil samples have been collected and analyzed by Region VII.

As dioxin contamination was found, the Region immediately proceeded with temporary measures to ensure that: (1) People would have very minimal contact, if any, with the dioxin-contaminated soil; (2) Barriers were put in place to prevent contact with or subsequent migration of the contaminated soils. Initially, families were relocated and at Times Beach and Minker the State purchased the properties using Superfund money; (3) EPA Region VII immediately began studies to remove and/or destroy the dioxin and at subsequent sites the dioxin-contaminated material was removed safely (gathered and contained) by cleaning and vacuuming.

The contaminated sites are located in two general areas of the State; Southwest Missouri, where the facility which was the origin of the waste is located, and the St. Louis area in Eastern Missouri. The sites are located in a wide variety of residential, industrial and rural settings, with diverse topographical and geological compositions. These variations combined with the massive volumes of contaminated materials (possibly more than two hundred thousand cubic yards) have posed a massive challenge to EPA and the state of Missouri.

However, cleanup projects are underway in Missouri. Cleanup activities have been safely completed at several sites and are well underway at a number of sites; still others are on the drawing board with mitigation plans moving ahead rapidly.

This emerging success story is due to the efforts and talents of several organizations. These include the EPA, The Centers for Disease Control, The National Institutes of Occupational Safety and Health, the Federal Emergency Management Agency, the Missouri Department of Natural Resources (MDNR), the Missouri Division of Health, and several contractors including Riedel International, IT Enviroscience, Roy F. Weston, Battelle-Northwest and CH_2M Hill in Eastern Missouri, and IT Enviroscience, Foster Wheeler Enviresponse and Cornejo and Sons in Southwest Missouri.

At sites in the St. Louis area where cleanup efforts are well underway the work is being carried out under hazard mitigation plans developed and peer reviewed as team efforts by the agencies and firms mentioned previously (1). These plans list as key components:

Community Relations - a comprehensive effort to ensure that affected residents, elected officials and the public in general are kept informed of all activities at the sites. Initiatives such as moving an incinerator to Southwest Missouri took a massive comprehensive effort to ensure that citizens received consistent, accurate technical information on a process with which they were unfamiliar. Components included briefings for neighbors of the Denney Farm and local elected officials. A special brochure and slide show detailed the burning process and fact sheets and photographs gave tangible evidence of what EPA planned to do. All major civic organizations were included in an intensive

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch015

speaking tour. Additionally, an EPA Information Center was established at the Denney Farm to answer questions posed by the public. A series of Information Center trailers at Castlewood, Quail Run, and Minker/ Stout in Eastern Missouri serve similar functions. Regular business hours are maintained, with EPA personnel also available on Saturday mornings (2).

Relocation - During the initial months of discovery of individual sites, there was no alternative other than possible relocation of some affected families to protect human health. As EPA has learned more about the characteristics of dioxin, mitigation is now possible through various alternatives, without long-term interruption of family life.

Cleanup, Excavation, Transportation and Storage - Based on health advisories from the Centers for Disease Control, and the state health agency, contaminated materials are excavated until no dioxin is left behind in the soil at a cleanup level of 1 ppb and, where excavation takes place, clean soil is put down so that people will not come in contact with dioxin-contaminated soil. The basic approach involves innovative excavation of 4- to 6-inch layers of contaminated material. Then the area is sampled in a statistically verifiable manner to assure cleanup below 1 ppb at the 95 percent confidence level. Excavated soil is loaded into specially designed, lined polypropylene containers and placed on plastic sheeting for subsequent loading by crane into lined trucks. Then loaded containers are transported to central storage facilities. Where excavation is completed that area is then filled back in with clean soil and revegetated.

These storage structures meet all pertinent requirements of the Resource Conservation and Recovery Act (RCRA). Each building has a concrete foundation and catch basin, a 1,000 psi rolled asphaltic concrete floor with sealer applied and each wood frame, steel-sided building encloses the entire foundation area. These buildings are secured with fences which will be posted, locked and inspected regularly by MDNR personnel. Thus, safe, secure, temporary storage is assured until a permanent disposal method can be planned and implemented.

All cleanup, transportation and storage procedures are conducted safely with methodologies to prevent fugitive emissions or spillage. A fundamental concept to any activity is: disturb the dioxin area only, preserving trees and other vegetation and the general environment wherever possible. The opened area is then restored with clean soil and a vegetation cover.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch015

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch015

Health and Safety - Plan prescribes the workplace practices and controls required to prevent employee exposure to 2,3,7,8-TCDD during handling of dioxin-contaminated materials.

Air Monitoring - An air sampling network for worker protection and general population exposure is established as part of cleanup activities. The network collects samples for the identification of total suspended solids and dioxin.

Contingency Plan - Outlines the procedures to be followed in the unlikely event of any unplanned release of hazardous materials at the site.

Structure Decontamination - Outlines a sampling plan for contaminated buildings. Subsequently describes cleanup procedures so that facility is cleaned until no dioxin is detected at analytical detection levels of 0.3 ppb or less.

Cost Summary - Outlines the budget for the project.

Concurrent with these efforts in Eastern Missouri, EPA embarked on an accelerated research and development program to find permanent solutions to the dioxin contamination problem. Paramount to this effort is a project whereby the Agency's mobile incinerator was transported from its home base in Edison, New Jersey, to the Denney Farm site in Southwest Missouri in December 1984.

The Denney Farm was selected for the incinerator because approximately 90 drums of waste containing dioxin from the production of hexachlorophene was buried in a trench on the farm. That material had been excavated and stored on site and was readily available for incineration. The initial mitigation of the site was performed by EPA. Syntex, the owner of the facility where the dioxin originated, is located only a few miles from the farm site and was willing to do the site preparation work plus provide other dioxin-contaminated materials for this research-oriented project.

The mobile incinerator passed a series of trial burns (conducted between February 25 and April 8, 1985) and attained destruction and removal efficiencies exceeding 99.999 percent as required by RCRA for 2,3,7,8-TCDD-contaminated materials burned in the system. Furthermore, the waste process streams, consisting of kiln ash and quench water, were successfully delisted.

Following the successful completion of the trial burns, an operating permit was issued for a full-field demonstration at the site. On July 23, 1985, this demonstration began by feeding dioxin-contaminated liquids and solids into the incinerator (3).

In addition to the Denney Farm wastes, four other dioxin sites in Southwest Missouri have been cleaned. The sites include Tally, Rusha, Erwin and Neosho. These cleanups were carried out using procedures similar to those previously described for sites in Eastern Missouri. The wastes from all the Southwest Missouri sites

In Solving Hazardous Waste Problems; Exner, J.;
ACS Symposium Series; American Chemical Society: Washington, DC, 1987.

were taken to Denney Farm for incineration. To date, more than two million pounds of solids and 180,000 pounds of liquids contaminated with 2,3,7,8-TCDD have been safely burned in the incinerator. The operation was temporarily halted awaiting additional Superfund dollars. Once burning resumes, only a few weeks are needed to complete the materials at the Denney Farm Site. During the interim period an engineering study has been conducted evaluating ways to increase the throughput and efficiency of the incinerator. Based on the results of this study, a decision was made to make modifications to the unit involving enlargement of the feed system, addition of a cyclone separator, replacing the particulate filter with a more efficient system and converting the air supply in the secondary combustion chamber to a pure oxygen system.

EPA is sensitive to the concerns of those residents whose communities have been contaminated with dioxin. We are moving forward with cleanup activities and tremendous progress has already been made toward our overall goal, which is a comprehensive, final solution to the Missouri dioxin problem.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch015

Literature Cited

(1) Keffer, W.G.; Wurtz, S.; Newbore, G.; Howard, D.; Exner, J.H.; "Quail Run Hazard Mitigation Plan," U.S. EPA Region 7 Report, Kansas City, KS, 1984.

(2) "Community Relations Plan, EPA Mobile Incinerator Project," U.S. Environmental Protection Agency, Office of Public Affairs, Region 7, May 1984.

(3) Proc. 1986 Haz. Materials Spills Conference St. Louis, Mo, May 1986.

RECEIVED November 25, 1986

Chapter 16

Case Study and Proposed Decontamination of a Closed Herbicide Plant in the Federal Republic of Germany

H.-J. Jürgens and R. Roth

Dekonta GmbH, Lotharstrasse 26, D-6500 Mainz, Federal Republic of Germany

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch016

This chapter describes the background to the closing of a $50 million chemical plant in Hamburg, Federal Republic of Germany, and proposed decontamination steps.

History

The plant, on the outskirts of Hamburg, began in 1923 as a branch for chemical pharmaceutical production of morphine and codeine. In 1951, production of herbicides began with the manufacture of hexachlorocyclohexane (HCH) and the corresponding isolation of lindane. The inactive isomers from this production were stored on site.

In 1953, recycling efforts for these waste isomers began, and thermal decomposition of these residues led to integration of several process steps (see figure). Thermal decomposition of the HCH isomers led to a 75% yield of 1,2,4-trichlorobenzene, which opened the process for production of 2,5-dichlorophenol and the corresponding bromination product. In addition to 2,5-dichloro-4-bromophenol production, the recycled trichlorobenzene was converted to 1,2,4,5-tetrachlorobenzene which could then be converted to 2,4,5-trichlorophenoxyacetic acid.

Formulation of insecticides and herbicides was carried out also.

The residues from the thermal decomposition were significantly smaller than the inactive HCH isomers. Although 2,3,7,8-TCDD content in 2,4,5-T production was known, the presence of polychlorinated dioxins in the residue from the thermal decomposition of HCH isomers was not demonstrated until 1984 (Table I).

0097-6156/87/0338-0221$06.00/0
© 1987 American Chemical Society

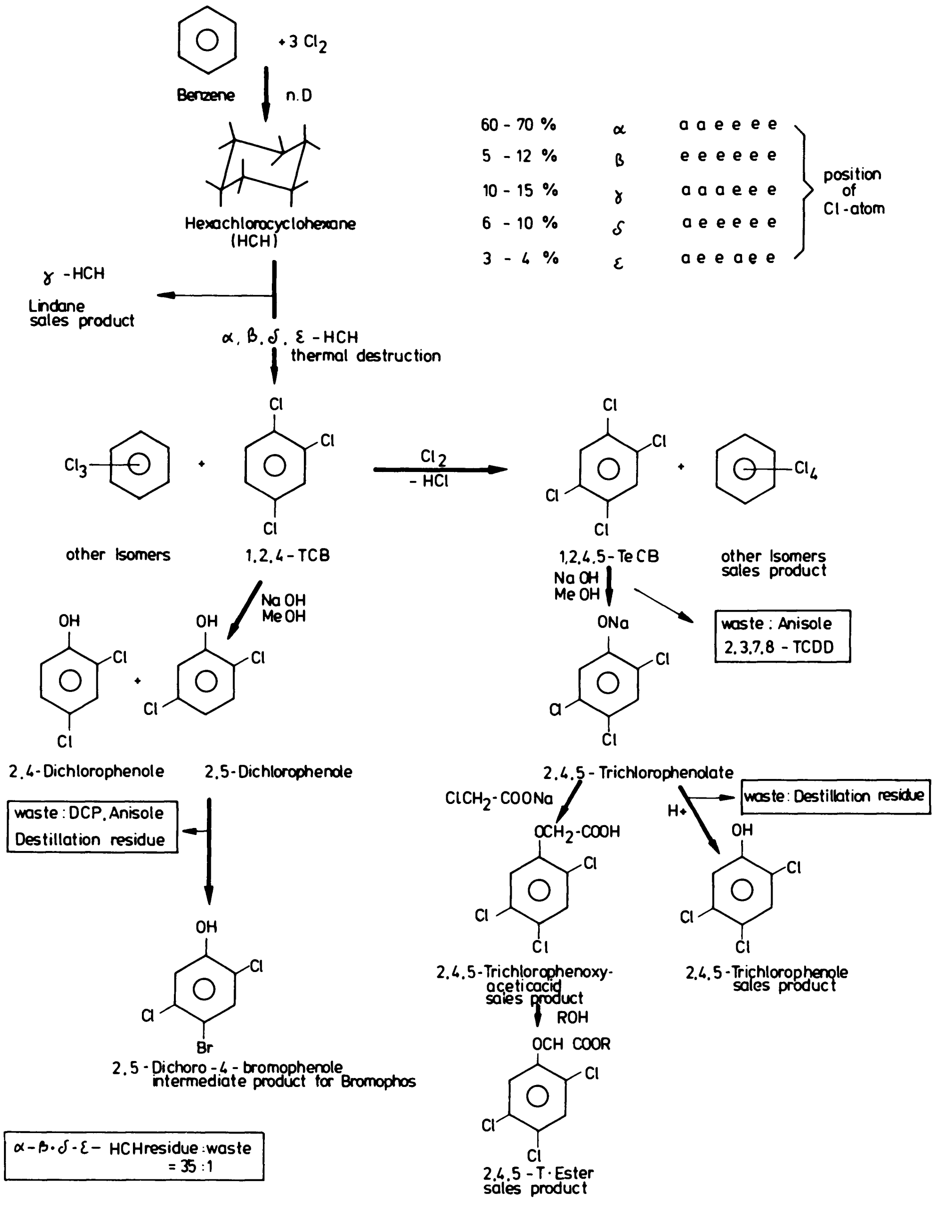


Figure 1. Recycling production of the Hamburg chemical plant.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch016

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch016

Table I: Analysis of the Thermal Destruction Residue of HCH (1)

Compound	Concentration (ppm)
2,3,7,8-TCDD	0.3
other isomers	12
1,2,3,7,8-Penta-CDD	7
other isomers	200
1,2,3,4,7,8-Hexa-CDD	45
1,2,3,6,7,8-	150
1,2,3,7,8,9-	65
other isomers	680
1,2,3,4,6,7,9-Hepta-CDD	1400
1,2,3,4,6,7,8-	3000
Octa-CDD	7600
2,3,7,8-TCDF	13
other isomers	10
1,2,3,7,8-Penta-CDF	1-2
2,3,4,7,8-	15
other isomers	40
1,2,3,4,7,8-Hexa-CDF	65
1,2,3,6,7,8-	17
2,3,4,6,7,8-	13
other isomers	230
1,2,3,4,6,7,8-Hepta-CDF	370
1,2,3,4,6,7,9-	15
1,2,3,4,6,8,9-	100
1,2,3,4,7,8,9-	10
Octa-CDF	200

Therefore, an exceptional recycling process, which converted waste HCH isomers to useful product, led to the closing of the lindane plant.

Plant Closing

How did it happen? The production facilities were not outmoded since the production plant was continually modernized according to best available technology, and between 1980 - 1984 a total of 30 million DM were invested in environmental protection.

A major reason for the closing was pressure by the public on government. The plant is separated by a road from a neighborhood with many small vegetable gardens. Increased air emissions of HCH, which led to increases of HCH in vegetables, gained extensive media attention in 1979, although the HCH content in vegetables was not demonstrated to be hazardous to health.

In 1981, a spectacular occupation of a stack in the plant by Greenpeace led to the beginning of the pressure by public interest groups and ecologists (Green Party) on the plant and on the appropriate agencies of the Free and Hanseatic City of Hamburg. In 1983, the attention resulting from the 41 drums of waste from Seveso, lost during transport, led to prohibition of transport of residues containing 2,3,7,8-TCDD in Germany. In this manner, incineration of the liquid residues at sea became eliminated as a disposal option. Consequently, the Hamburg-Moorfleet plant stopped production of 2,4,5-T, for which a new building and process had been erected in 1978. The newly founded department of environmental protection in Hamburg increased its search for dioxin in production, waste, aqueous discharges, and in soil. After soil from the plant was found to contain about 30 ppb of 2,3,7,8-TCDD, and because of the known TCDD contents in residues from 2,4,5-T production, the company remained in the headlines.

References to the company appeared in almost every publication relating to TCDD, either municipal incinerators, landfills, or residues in fish. When it was demonstrated that the residue from the thermal decomposition of HCH contained, unexpectedly, 2,3,7,8-TCDD and other polychlorinated dioxins and furans, the company was required by the Hamburg environmental protection department to meet the following effluent limits: TCDD contents in residue 1 μg/kg, in air 1 pg/m^3 and in aqueous discharge 1 ng/l (2). These limits, which had been suggested by Epstein and others at the Georgswerder-Symposium, in June 1984, were opposed by the company but upheld by the Hamburg courts. Since these limits represented analytical detection limits in Germany, and since future lowering of the detection levels was probable, it was uncertain whether these values could ever be met. Consequently, production was stopped at the site. For comparison, at the same time the allowed emissions from two Hamburg municipal incinerators were 200 pg/m^3 and 650 pg/m^3 (3).

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch016

Cleanup Plans

Dekonta is engaged in planning and carrying out appropriate measures for converting the plant site into a reusable industrial site. The project is divided into two parts, decontamination of soil and underlying areas, and cleaning and dismantling of buildings and equipment.

We are operating under the following guidelines:

- Liquid wastes containing dioxins are not allowed in normal landfills.

- Incineration in land-based facilities that are capable of destroying dioxins is not allowed by local authorities.

- Incineration at sea also cannot be permitted.

- If permits for disposal in a secure landfill could be obtained, transportation of wastes containing dioxin above 10 µg/kg is prohibited. Exceptions to this transportation prohibition are possible but not specified. Therefore, on-site destruction of liquid wastes remains the only alternative, however, specific procedures are not defined.

- Solid wastes containing dioxins can be disposed in one secure landfill, an old salt mine located at Herfa-Neurode in Hessen. There are three major restrictions:

  1. Only wastes from the Federal Republic of Germany are accepted.

  2. The landfill imposes special packaging restrictions which, in case of our wastes, double the disposal weight.

  3. The possible exception of the transportation prohibition is tied to specific packaging requirements. A distinction is made between those wastes containing greater than 10 µg/kg and those below that number.

Soil and Groundwater

The 80,000 sq meter area has been divided into a grid and, since May 1985, 46 exploratory drillings to 30 to 60 meters and 118 drillings to 8 meter depths have been carried out. In addition, 32 wells are being drilled to 30 meter depth in the immediate vicinity of the plant site. The soil samples are being analysed for total extractable halide, specific production materials by gas chromatography,

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch016

and total PCDD's. The geological and hydrogeolocial knowledge from this investigation, coupled with the analytical results, will be the basis for a decontamination plan.

Preliminary results from the exploratory borings are:

- The plant area contains areas that are highly contaminated, with EOX values up to 284,000 ppm.
- The pollution result from chlorobenzenes, HCH, chlorophenols and 2,4,5-T-acid.
- A few samples from the surface area contain 2,3,7,8-TCDD. One sample contains 9 ppm.
- Groundwater below contaminated soil is also contaminated, 2,3,7,8-TCDD has been not detected.
- Contamination decreases markedly outside the plant. EOX-values in soil are generally less than 20 ppm.
- Geologic conditions limit the groundwater contamination to a reasonably well-defined plume towards the South.

14% of the borings at depth contain EOX at greater than 20 ppm, whereas 40% of the shallow borings have similar contamination levels.

Soil contaminated with levels of TCDD up to 9 ppm are subject to immediate response. About 2,000 m^3 of such soil will be excavated and placed into an interim storage area upon approval by the Hamburg authorities. These contaminated soils will be stored until a destruction method becomes available. Such a method will be proposed upon conclusion of the drilling test program. Soil decontamination methods must also take account of the building and equipment cleanup procedures.

Buildings and Equipment

Equipment is to be cleaned for off-site reuse or scrapping. Buildings are to be cleaned or demolished.

The degree of contamination of the buildings and the equipment is being determined according to the principle of worst case. Selection of samples from areas most likely to be contaminated will lead to the establishment of personal protection measures and to the appropriate decontamination method. Four levels of worker protection have been established with appropriate protective equipment:

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch016

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch016

0 no chemical contamination
ea requires normal work clothing.

A Chemical contamination and 2,3,7,8-TCDD equivalents $<$ 0,01 ppm
Work in this area will be carried out in coated Tyvex, safety shoes with booties, chemically resistant gloves, partial face mask with particulate and organic vapor respirators, and safety glasses.

B Chemical contamination and 2,3,7,8-TCDD equivalents between 0,01 - 1,0 ppm
In this area, the area A procedures will be expanded to include full face mask and more frequent clothing changes.

C 2,3,7,8-TCDD equivalents greater than 1 ppm. Work will be carried out in air-supplied suits.

Workers in the project are being monitored by a comprehensive occupational health examination on a 6-month-basis.

A comprehensive sample tracking procedure has been developed. For example, the computerised system assigns a number which contains a sequential sample number, day and date, coordinates according to a site grid (length, width, level), building number, and sample type such as:

Sequential sample number	Day+Date	Grid-Coordinates			building number	sample typ
200002	MI 13.02.85	L 272	B 102	H 4,6	B250	02

Additional information is added to define the specific piece of equipment and sample location. This procedure will also contain information about decontamination procedures, demolition, disposition of residues, and disposition of cleaned equipment for reuse or scrapping.

Target levels were established for cleaning equipment surfaces. These levels for normal products were proposed based on a simple evaluation of toxicological data, such as ADI (allowable daily intake) or LD_{50}, and estimates of dermal contact and potential skin adsorption (Table II).

Table II. TARGET LEVELS FOR EQUIPMENT DECONTAMINATION

Compound	Concentration mg/m^2	
HCH	18 -	77
1,2,4-Trichlorobenzene	2000 -	8800
2,4,5-T-acid	107 -	462
2,4,5-Trichlorophenol	89 -	385
2,3,7,8-TCDD	0,000004 -	0,000155

Cleaning and dismantling of the lindane plant will begin this summer. Although this unit contains no PCDD, worker protection and decontamination procedures will be established as training for cleanup of PCDD-containing plants. Cleaning water and surface runoff will be collected and treated by filtration through an activated carbon system that is being constructed by Dekonta. We expect to meet or exceed the discharge criteria to the Hamburg waste water treatment plant of 1 ng/l of TCDD.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch016

Literature Cited

1) Analysis was made by:

Dr. H.R. Buser, Eidgenössische Forschungsanstalt, CH-8820 Wädenswil, Schweiz

2) Freie und Hansestadt Hamburg
Behörde für Bezirksangelegenheiten, Naturschutz und Umweltgestaltung
-Amt für Umweltschutz-
Nachträgliche Anordnung nach § 17 BImSchG zu den Genehmigungsbescheiden der Fa. C.H. Boehringer Sohn vom 18.06.1984

3) Bürgerschaft der Freien und Hansestadt Hamburg
Drucksache 11/3159 vom 30.10.1984
Mitteilung des Senats an die Bürgerschaft

RECEIVED November 25, 1986

Chapter 17

Military Sites Contaminated with 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin

Permitting Remedial Action Research

Judy N. Casanova[1] **and Robert F. Olfenbuttel**[2]

[1]EG&G Idaho, P.O. Box 1625, Idaho Falls, ID 83415
[2]Headquarters, Air Force Engineering Services Center, Research & Development Directorate, Environics Division, Tyndall Air Force Base, FL 32403

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch017

Recent amendments to the 1984 Hazardous and Solid Waste Act have discouraged the disposal of dioxin-containing wastes on land and encouraged the development of unique treatment technologies. The 1984 Amendments make provisions for the waiver of research development and demonstration (RD&D) permit requirements by Environmental Protection Agency regional administrators to facilitate and expedite critically needed research (pilot-scale and prototype testing). Although these regulations have been established, they have done little to speed up the permitting process. Problems and costs of RD&D permitting are discussed, together with the related issue of hazardous waste delisting. Recommendations are made to expedite the RD&D process and meet the intent of the 1984 Amendments.

The Air Force Engineering and Services Center, located at Tyndall Air Force Base, Florida, has the lead responsibility within the Air Force for conducting research and development (R&D) in a wide spectrum of environmental problem areas. Chief among these challenges is hazardous waste reduction, control, and site cleanup.

The R&D program has two thrusts: 1) Basic research and laboratory studies to establish the technical base from which innovative and cost-effective hazardous waste site cleanup technologies can be developed, and 2) field testing of promising technologies for which no performance "track record" is available. The latter includes pilot and prototype-scale demonstrations, and is the focus of this paper as it applies to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD).

Identifying candidate innovative and alternative technologies for dioxin-contaminated soil cleanup has not been a difficult process. Many organizations, ranging from small entrepreneurial individuals and businesses to established waste management firms, have unique approaches and ideas for solving contamination problems. The challenge has not been in selecting the most promising technologies, but in conducting field tests and evaluations (T&E)

0097-6156/87/0338-0229$06.00/0
© 1987 American Chemical Society

in a reasonable, timely fashion. Unfortunately, the current regulatory permitting process is inconsistent, often costly, and time consuming.

This paper discusses, through case studies, the U.S. Air Force's (USAF) experiences with the activities, time, and costs involved in meeting regulatory requirements to conduct small- and large-scale technology demonstration projects under two different governing acts and four different programs. The analysis is limited to those activities that were part of the hazardous waste permitting process. The results indicate a lack of economic considerations in the current regulatory program for research, development and demonstration (RD&D) projects for hazardous waste management.

The basic goal of environmental laws is to induce people to withdraw, use, reuse, and discharge materials in amounts and ways that take proper account of the damages that discharges can do to human welfare(1). This also includes taking care of past problems in a manner that will prevent further damage to the environment and human health. The inefficiencies and costs currently encountered in an effort to correct an environmental problem act as disincentives for commercial development and application of new and better technologies to remedy environmental problems. Furthermore, they retard rather than expedite technology demonstration and site rehabilitation.

Background

During the Vietnam conflict of the 1960s and 1970s, the Department of Defense (DOD) launched a massive defoliation program to eliminate the vegetation used by the enemy for concealment. A herbicide commonly known as Agent Orange was used for this program in which 11 million gallons were sprayed over 0.5 million acres of Vietnam between 1965 and 1970.

Agent Orange was formulated to contain a 50:50 mixture of two active ingredients: 2,4-D (2,4-dichlorophenoxyacetic acid) and 2,4,5-T (2,4,5-trichlorophenoxyacetic acid). In 1970, studies were published which indicated that 2,4,5-T was a teratogen (a substance which causes defects of fetal development)(2). Subsequent studies revealed that the teratogenic effects resulted from a toxic contaminant which was inadvertently formed in the production of 2,4,5-T. This substance was identified as TCDD.

In 1970, the Secretaries of Agriculture; Health, Education, and Welfare; and the Interior jointly announced the suspension of certain uses of 2,4,5-T. Subsequently, the DOD suspended use of Agent Orange. At the time of the suspension, the USAF had a 0.85 million gallon inventory of the herbicide at the Naval Construction Battalion Center (NCBC) in Gulfport, Mississippi. The 1.37 million gallons in South Vietnam were moved to Johnston Island, Pacific Ocean, in 1972.

In 1977, the USAF disposed of the entire stockpile of Agent Orange by high-temperature incineration at sea. The disposal project was accomplished according to requirements of the U.S. Environmental Protection Agency (EPA) under ocean-dumping requirements promulgated under the Federal Water Pollution Control Act. The approximate cost of disposal was $70 million(3).

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch017

At this time, regulation of dioxin was piecemeal and fragmented between authorities who were functioning under the Insecticide Act of 1910, the Federal Insecticide, Fungicide and Rodenticide Act of 1972, the Federal Water Pollution Control Act as amended by the Clean Water Act of 1977, the Clean Air Act as amended, the Occupational Safety and Health Act, the Consumer Product Safety Act, and the Toxic Substances Control Act of 1976 (TSCA).

Following destruction of the Agent Orange in 1977, the USAF committed to EPA a program of follow-up storage site reclamation and environmental monitoring. (Due to leakage during storage or spillage from handling, the old storage sites were contaminated with undetermined quantities of Agent Orange.) In an attempt to restore the storage sites to beneficial use, the USAF entered programs which included the following:

1. Conducting sampling and analysis programs to determine the vertical and horizontal extent of contamination of the old storage sites at Johnston Island and NCBC.
2. Selecting, field testing, demonstrating, and evaluating different dioxin destruction technologies.
3. Determining the best technology to be used for eventual cleanup of dioxin-contaminated sites.

RD&D Experiences

Small-Scale Demonstrations Under TSCA. As a result of a competitive procurement, the USAF selected two promising technologies for test and evaluation at the dioxin-contaminated site on the NCBC, in Gulfport, Mississippi. The site is a USAF responsibility. These two technologies were a thermal desorption/U.V. destruction system developed by International Technologies, Inc. (IT Corporation), and an advanced electric reactor developed by the J.M. Huber Corporation. In early 1985, these two technologies were tested at the NCBC, governed by regulations promulgated under the Toxic Substances Control Act (TSCA), under the authority of the EPA's Dioxin Disposal Advisory Group. To meet the regulatory requirements for this effort, a document was assembled which explained the purpose of the project, description of the two technologies to be tested, and plans for protection of human health and the environment.

As indicated in Table I, it took a little more than two months to get the necessary approvals for conducting the experiments. Total cost for planning, preparing, and obtaining the approvals was approximately $60,000.

Resource Conservation and Recovery Act of 1976 (RCRA) as amended by the Hazardous and Solid Waste Amendments of 1984 (HSWA). In 1976, RCRA was passed to provide a multifaceted approach toward solving problems caused by solid waste. Salient aspects of RCRA were the expansion of the role of the federal government in the field of solid waste disposal management with particular emphasis on regulation of hazardous wastes(4).

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch017

TABLE I. Permitting Costs for Small-Scale Demonstrations (NCBC)

Governing Act: Toxic Substances Control Act

Regulatory Authority: EPA Dioxin Disposal Advisory Group
EPA Region IV Office

Activity	Approx. Cost*
Planning Meetings	$ 9,712
Preparing Document	34,000
Preparing Presentations	2,000
Conducting 3 Presentations	8,500
Lag Times: The activities for obtaining approvals were initiated in March 1985 and the demonstrations began in May 1985, resulting in a lag-time equipment lease cost of $6,000.	6,000
Total Cost:	$60,212

*Cost estimates include labor, G&A, overhead, materials, and travel costs. They do not include contractor fees, which were not applicable.

In 1984, the President signed HWSA into law. The Amendments made many changes to the existing EPA hazardous waste management program developed under the Resource Conservation and Recovery Act (RCRA) of 1976. Significantly, the Amendments reflect the intent of Congress to encourage innovative research and development of advanced technology for destroying hazardous wastes. Section 3005(g) gives EPA the authority to issue research, development and demonstration (RD&D) permits, without promulgation of permitting regulations (under Part 264) that would establish standards for technologies or processes that treat hazardous waste in an innovative and experimental manner. In addition, the EPA may modify or waive the permitting and technical requirements applicable to other types of hazardous waste management facilities as long as: 1) human health and the environment are protected; 2) financial responsibilities are maintained; and 3) public participation requirements are fully adhered to.

In 1985, management of dioxins was transferred from TSCA to authority under RCRA, as amended by HSWA. The new Amendments also added paragraph 3001(f), establishing specific criteria and procedures for delisting petitions. In addition, permitting authority was delegated to the Regional EPA Offices.

As codified in 40 CFR 270.65, these RD&D permits were conceived to aid in the development of safe alternatives to land disposal of hazardous waste, a primary goal of the Amendments, by expediting the permitting process to demonstrate the technical and/or economic feasibility of experimental and innovative technologies and processes. Indeed, the EPA Director of the Office of Solid Waste stated that processing RD&D permits is "one of our highest

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch017

priorities, as indicated in both the National Permits Strategy and the RCRA Implementation Plan for FY86"(5).

The goal of expediting everything, however, has not been realized in a consistent manner. The USAF changed its program to comply with these regulations and to continue with testing and demonstrating technologies that would develop the most effective and cost-beneficial method to clean up the dioxin contamination. This resulted in inconsistent response among regional EPA offices, extraordinary delays in permitting some activities, and concomitant high costs which threaten the viability of some projects. The problems described below also apply to the EPA R&D community as well, and must be resolved if the intent of Congress is to be realized and the challenge of contaminated sites restoration is to be effectively met.

Case Study 1: A Small-Scale Demonstration Under RCRA. As discussed previously, two technologies were tested and evaluated at the NCBC, Mississippi in mid-1985. The approval process went smoothly and quickly under TSCA. Preliminary data indicate that the systems were capable of reducing the level of dioxin in soil to less than 1 part per billion.

The apparent success of the systems led to a decision to continue testing the IT Corporation technology at Johnston Island, in the Pacific Ocean, as originally planned. The Johnston Island site offered a different soil matrix and further opportunity to characterize the maintainability and reliability of the system.

Permitting of this single technology demonstration fell under the jurisdiction of EPA Region IX and the RCRA system. Table II presents the activities and costs to date (September 1986) under RCRA requirements. The following discussion refers to activities listed in Table II and is instructive with regard to requirements and difficulties.

1. (See 1, Table II) Under RCRA, generators of hazardous waste must obtain an identification number from the appropriate EPA Regional Office (EPA Form 3510-1).

2. (See 2, Table III) Under RCRA, certain projects can be designated as RD&D projects (Hazardous and Solid Waste Amendments of 1984). Specific requirements to obtain a permit for an RD&D project are up to the discretion of each EPA region. Therefore, requirements can vary greatly from region to region, or where applicable, from state to state.

3. (See 4, Table II) Each project is assigned a permit writer in the appropriate EPA Regional Office. For an RD&D permit, EPA is not required to respond within legal time limits. Because of the time constraints placed on the assigned permit writer, and the low priority given RD&D projects in this region, it became necessary to use a large portion of the budget for travel in order to meet with the permit writer in efforts to expedite the permit process.

4. (See 6, Table II) This activity is the result of regulatory complications. EPA published regulations in 1985(6) which specifically required that dioxin-contaminated soil which had been thermally treated to remove dioxin would still be considered a hazardous waste (F027 and F028 wastes). This meant that the treated soil would have to be placed into appropriate containers and sent to

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch017

TABLE II. Permitting Costs for Small-Scale Demo (Johnston Island)

Governing Act: Resource Conservation & Recovery Act, (as amended)

Regulatory Authority: EPA Regional Office

Activity	Approx. Cost*
1. Submit application for notification of Hazardous Waste Activity	$ 2,000
2. Assemble and submit Research, Development, and Demonstration permit application	17,500
3. Obtain EPA comments	2,000
4. Respond to EPA comments/requests	17,250
5. Revise and resubmit permit application	2,000
6. Submit application for waste storage facility	300
7. Respond to Freedom of Information (FOI) request	1,000
8. Respond to additional EPA comments/requests	6,250
9. Revise and resubmit permit application	1,000
10. Resubmit Notification of Hazardous Waste Activity and interim storage application forms	500
11. Efforts expended to expedite permit issuance	2,000
12. Meet with Region IX to discuss draft permit	5,000
13. Revise and resubmit permit application	1,000
14. Participate in public hearings	24,500
15. Respond to Regional Counsel on FOI request	4,300
16. Time lags: The permitting activities listed above were begun in April 1985. The start date will be June 1986. Thus, costs to transport equipment, associated equipment rental, and additional costs incurred to reschedule and replan were $48,080.	48,080
Total Cost:	$134,680

*Cost estimates include labor, G&A, overhead, materials, and travel costs. They do not include contractor fees, which were not applicable.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch017

TABLE III: Permitting Costs for a Full-Scale Demonstration

Governing Act: Resource Conservation and Recovery Act of 1976 (as amended)

Regulatory Authority: EPA Region IV Office
EPA Headquarters, Office of Solid Waste
Mississippi State Bureau of Pollution Control

Activity	Approx. Cost*
1. Planning Meetings for RD&D permit	$ 19,325
2. Meeting to initiate delisting activities	11,325
3. Preparation of RD&D permit application	22,500
4. Preparation of Delisting Petition form	25,000
5. Meet with State officials	4,500
6. Meet with EPA Regional officials	4,500
7. Meet with EPA Headquarters officials	4,500
8. Submit Notification of Hazardous Waste Activity	1,000
9. Prepare Environmental Assessment	10,000
10. Resolve EPA and State comments	4,250
11. Revise and resubmit permit application	2,000
12. Support public information activities	8,382
13. Continue resolution of issues and submission of information for delisting process	25,000
14. Incorporate delisting criteria into RD&D test plans	7,000
15. Lag time	
Total Cost:	$149,282

*Cost estimates include labor, G&A, overhead, materials, and travel costs. They do not include contractor fees, which were not applicable.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch017

permitted hazardous waste disposal facilities for final disposal. These rules create the following problems:

a. Although EPA has published rules to allow the disposal of dioxin-containing waste at interim status facilities, to our knowledge there are no disposal facilities which are currently accepting such waste. This creates another permitting impass.

b. Without the ability to dispose of treated soil, a storage permit must be obtained to allow for storage of the treated soil until an appropriate disposal facility is established or until regulations are adjusted. EPA Form 3510 was submitted to Region IX.

Because no "de minimus" limit has been established as to when something contaminated with dioxins is considered "clean," there is no method to prove the material has been treated to an acceptable limit. Therefore, there is no economic benefit to clean up a large quantity of soil contaminated with F027 wastes if it must be managed as a hazardous waste after treatment. The only recourse available is to pursue "delisting" of the treated soil through EPA Headquarters, Office of Solid Waste - that is, to go through a petitioning process in which one attempts to prove to EPA that the soil can be placed back on the ground without harm to human health or the environment.

c. The delisting process usually takes a minimum of one year to complete. As of September 1986, EPA had approved nine delisting petitions, including the EPA's Mobile Incineration System use at Denney Farms in McDowell, Missouri(7).

At the moment, an RD&D permit must be obtained to legally perform this technology demonstration. In order to perform the demonstration, a storage permit must be obtained to legally store wastes until the wastes can be legally disposed of or legally delisted or until the regulations are changed, whichever comes first.

5. (See 7, Table II) A copy of the USAF RD&D permit application was requested from the EPA by another party under the Freedom of Information (FOI) Act. To protect confidential process information, a Confidentiality Claim was filed with Region IX.

6. (See 8,9, Table II) Five months after the resubmission of the permit application, the EPA Regional Office submitted a second set of comments and requirements. The permit application was revised and submitted a third time.

7. (See 10, Table II) The application forms from Items 1 and 6 could not be located at the Regional Office. Both were resubmitted.

8. (See 13, Table II) Following the meeting with Region IX to discuss the draft permit, it was agreed that the permit application would be revised and resubmitted; and that another version with the revisions and confidential information blacked out would also be submitted.

9. (See 15, Table II) In June 1986, notification was received that the response to the FOI request submitted in August 1985 had been submitted for review to the Regional Counsel's Office. Considerable effort was expended responding to questions and reevaluating information to obtain approval for the "confidentiality" claim.

10. (See 16, Table II) After review of the second submittal of the permit application, a draft permit was estimated to be

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch017

forthcoming in October 1985. Because equipment transport to Johnston Island takes about one month, the equipment was en route to the project site before the numerous delays occurred; therefore, equipment rental costs have been incurred since September 1985. Since additional transport costs have been incurred because of the significant permit process delay, these additional costs are included in the permit process.

Discussion

The total costs in Tables I and II may be misleading in that Table I is the total cost for conducting two technology demonstrations, and the total costs in Table II are for only one demonstration. If the regulatory programs had been somewhat consistent, one could expect the costs in Table II to be about one-half of those in Table I. However, the costs are 2.2 times higher.

The goal of such regulatory programs is to protect human health and the environment. Because we believe that efforts to protect human health and the environment were equivalent under both the TSCA and RCRA demonstrations, it does not appear that accomplishing this goal is more effective under RCRA than TSCA.

Additionally, based on the preceding data, the goals of the RD&D permit process, which are to expedite the development of land disposal alternatives and simplify the permit application and issuance process, are not being met. Not only are the goals not being met, it would seem fair to say that the complexity of the present hazardous waste management system is promoting economic inefficiencies which cannot be justified.

Case Study 2: Full-Scale Demonstration. A third technology demonstration project is currently being pursued. Although similar in nature to the previously mentioned demonstrations, different regulatory problems are being encountered with this RD&D project.

Thermal treatment appears to be the most readily available remedial action technology for dioxin. However, no data are available to confirm the maintainability, reliability and cost-effectiveness of transportable commercial systems. Through a competitive process, the USAF selected ENSCO Corporation for a full-scale field test of their rotary-kiln combustor. The test will be conducted at the NCBC, Mississippi and will involve treatment of approximately 9000 cubic yards of contaminated soil. An intense analytical program and engineering failure mode analysis will support the project. Testing is planned for November 1986 - March 1987.

Permitting of this single technology demonstration fell under the jurisdiction of EPA Region IV, whose staff has been outstanding in attitude and actions. Table III summarizes the actitivies and costs as of September 1986.

The following provides additional information on selected activities listed in Table III.

1. (See 1, Table III) The cost for the planning meetings is somewhat misleading in that the original planning meetings took place on Johnston Island, where the full-scale demonstration was originally planned to take place. However, because of the problems already encountered, and the associated costs with Region IX and the

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch017

higher costs of conducting a demonstration on Johnston Island, the demonstration was replanned for NCBC.

2. (See 2, Table III) As mentioned in Table II, dioxin-contaminated soil which has undergone thermal treatment must still be managed as a hazardous waste (i.e., properly disposed of at a permitted hazardous waste facility). The only alternative available for this project was to petition EPA Headquarters, Office of Solid Waste for delisting of the treated soil. Because the draft guidance manual for RD&D Permits places a ceiling on the maximum amount of hazardous waste stored at any time for experimental purposes, obtaining a storage permit pending delisting would not be useful; therefore, delisting must be obtained. However, to follow delisting procedures, test burns of the machine and feedstock must be conducted and data from those tests evaluated before issuance of delisting. This would mean a downtime of several months from test burn to official delisting.

3. (See 9, Table III) Because of the size of the full-scale demonstration project, an Environmental Assessment was prepared to fulfill National Environmental Policy Act requirements.

4. (See 15, Table III) The RD&D permit application was submitted to Region IV and the State of Mississippi on January 21, 1986. The final permit was received July 2, 1986. Lag-time costs will be incurred for this project. However, these costs will be a result of the delisting process, rather than the RD&D permit process.

Table IV compares the costs and time involved in the RD&D permitting process in two different EPA regions.

TABLE IV. PERMITTING PROCESS COMPARISON

	Small Scale Demo Region IX	Large Scale Demo Region IV
Permit application (original)	$17,500	$22,500
Comment resolution	19,250	4,250
Resubmission	2,000	2,000
Comment resolution	6,250	-----
Resubmission	1,000	-----
Comment resolution	5,000	-----
Resubmission	1,000	-----
(does not include public hearing process)	$52,000	$28,750
Notification of Hazardous Waste Activity)	2,000	1,000
Resubmittal	250	-----
TOTAL	$54,250	$29,750
TIME:	13 Months	5 Months

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch017

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch017

Delisting: A Serious Issue Requiring Change

Researchers face significant potential time delays and costs associated with "delisting" the output of the treatment systems. As already mentioned, HSWA added paragraph (f) to section 3001 establishing specific criteria and procedures for delisting petitions. This requires the EPA to consider additional factors, such as constituents other than those for which the waste was listed, if the Agency believes there is a reasonable likelihood that the additional factors could cause the waste to be hazardous.

Although the delisting petition process was established around regular RCRA treatment operations and/or production facilities, it is presently being applied to RD&D with adverse consequences. Delisting is of particular concern because we are dealing with: 1) soils containing listed F027 substances and constituents which are identified as acutely hazardous wastes; and 2) the treated soil resulting from the demonstration which is specifically listed as an F028 waste, i.e., residues from the incineration or thermal treatment of soil contaminated with dioxin(6).

The rationale for requiring delisting is reasonable in relation to regular treatment operations. However, for RD&D the process must be streamlined and the risk for investors reduced. Currently, a demonstration project must be stopped soon after it has started in order to submit laboratory data on the products of the treatment process, via a formal "delisting petition" to HQ EPA. The downtime engendered by this sequence is expensive, particularly when it involves a full-scale incinerator at a cost of thousands of dollars per day. Added to this is the uncertainty of criteria for the delisting and the fact that EPA has up to 24 months to grant or deny the petition. Taken together, these factors create the potential for pushing the proposed demonstration beyond acceptable cost risk. It will take very few experiences of this type to discourage further contributions to technology development by investors both within government agencies and the entrepreneurs upon whom society depends for innovative ideas.

The issue of delisting is currently being considered on a specific and generic basis by legal counsel and the Waste Identification Branch at HQ EPA. Delisting could be expedited by the following suggested process: EPA establish a delisting standard in advance of the demonstration start. This standard can be based on laboratory and pilot scale data. The delisting standard would be published in the Federal Register and subject to a formal public comment period. The EPA reviews the comments and issues a formal delisting action that would be contingent on the petitioner demonstrating that the treated soil did, in fact, meet the delisting standard. This scenario would minimize the downtime of equipment at the demonstration site since the formal process involved with delisting would be completed prior to project start. Verification of waste residues' quality would be the only step necessary and should be accomplished rapidly by EPA.

As of September 1986, EPA's guidance on delisting was contained in EPA publication 530-SW-85-003(8).

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch017

Contents of RD&D Permit Applications

When the permit application was submitted to Region IX, guidance did not exist on contents of an RD&D permit application. For the purpose of the first technology demonstrations, an outline was submitted to EPA's Dioxin Disposal Advisory Group (DDAG) for the proposed contents. This outline was acceptable to both DDAG and the State of Mississippi, and was therefore used as a guide for the contents of the RD&D permit application to Region IX.

This resulted in some problems with the EPA. EPA believed that some items in the application were extraneous, while others were omitted. Because reviews were performed by different offices within EPA and at different times, several rounds of comments required resolution.

Table V presents the major sections from the Table of Contents of the USAF RD&D permit applications. It contains all the significant information required by the draft guidance document for RD&D permits issued from EPA Headquarters(5).

The most recent permit guidance (September 1986) is contained in Office of Solid Waste and Emergency Response (OSWER) Policy Directive No. 9527.00-1A(9).

TABLE V. Major Headings for Contents of RD&D Permit Application

Executive Summary
Introduction and Background (includes site description)
Process Description (includes process monitoring, inspection and maintenance, and test plan)
Process Validation (includes technology assessment and process confirmation)
Emergency and Contingency Plan
Health and Environmental Protection
Monitoring Plans (includes independent verification and QA/QC plans)
Financial Responsibility
Closure and Equipment Decontamination Plan
Public Relations
Personnel Qualifications
Appendices Sampling and Analysis Matrix and Assessment of Part 264 requirements

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch017

Conclusions

The difficulties experienced in seeking RD&D permits are related to a number of factors. The EPA Administrator has delegated permit authority to each of the Regional Administrators. However, a demonstrated inconsistency among these authorities and/or their staffs regarding their willingness to issue or waive permits indicates that regional sensitivities are sufficiently strong to override the national intent of the HSWA legislation. It also indicates that guidance from HQ EPA is not firm enough to foster an equal opportunity for obtaining permits around the country.

In addition, it appears that the size and capabilities of some staffs are insufficient to handle the permit process in a timely manner. To apparently mitigate this difficulty, HQ EPA has arranged for assistance from both the Alternative Technologies Division at the Office of Research and Development in Cinncinnati, Ohio and the Headquarters Permit Assistance Team (PAT). Although these resources add expertise to permit application reviews, they also can add time to a process which is already too long. As discussed in these case studies, the turnaround time from original application to issuance of a draft permit in Region IX took 13 months. Contrast this with the office in Region IV which had the willingness and staff capability to process and award a permit in 150 days.

The expense involved in waiting for permit application approval is significant; in addition, the wait endangers the availability of funds for the project and creates a veritable logjam of other hazardous waste treatment projects scheduled for test and evaluation.

The major problem is that broad preliminary requirements were issued in the beginning. The dioxin research program was developed in a "crisis" atmosphere, resulting "in instability and some hasty decisions that, in hindsight, may need to be changed."(10,11) In a similar way, the regulated community's approach to dioxin management was developed under a framework of "hurry-up" legislation. In attempting to live with these regulations, researchers find that they have been written into a corner shared by authorities who are also struggling for guidance and well-defined direction. Unless effective changes are made to the permitting and delisting process, not only will we find it difficult to meet our own environmental cleanup requirements, but such major programs as EPA's proposed Superfund Innovative Technology Evaluation (SITE) may never get off the ground.

Because of the rapidly evolving hazardous waste regulations, it is difficult if not impossible, to predict the effects each new rule or promulgation of regulations will have on a particular project. Dioxins and similar compounds have been hit especially hard by several iterations of new rulemaking. Perhaps the best lesson to be learned from the subject technology demonstrations is that a very careful assessment of the regulatory authorities involved should be a first step in initiating a new project. Personnel involved, their willingness to work with people, and general policy and atmosphere of the Region and/or State involved can be a very good indicator of the potential of overcoming regulatory problems which, at this time, determine whether or not a project can occur.

In Solving Hazardous Waste Problems; Exner, J.;
ACS Symposium Series; American Chemical Society: Washington, DC, 1987.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch017

Recommendations

The difficulties discussed in this paper are certainly the product of an immature program and participants must provide constructive recommendations to improve the system wherever possible. Therefore, the following recommendations are offered to EPA for promoting RD&D projects:

1. Centralize RD&D permitting at HQ EPA Office of Research and Development (EPA/ORD); or adopt a mandatory guideline for regional RD&D permitting.

2. Modify the RCRA regulations to accommodate timely issuance of RD&D permits including a shortened timeframe for final permit award (e.g., 90-120 days).

3. Modify RD&D permit requirements to allow federal agencies to conduct RD&D on federal property. The federal agencies would comply with the "intent" of RCRA without participating in the formal permitting process.

4. Modify "delisting" procedures and criteria to expedite the RD&D process.

Centralization of RD&D permitting at EPA/ORD would add a national perspective to the RD&D permitting process, something that is currently missing. Additionally, shifting the permitting to HQ EPA would eliminate much of the regional sensitivities and politics. Realistically speaking, this approach has little chance of adoption.

The adoption of a mandatory permitting suspense of 90-120 days would greatly shorten the time required in some regions to obtain an RD&D permit. Adequate staffing must be provided to accomplish this option. Federal agencies, including EPA's own research groups, should be allowed to conduct timely, problem solving research and development on federally owned facilities without the need for formal RCRA permitting. The federal agencies should only be tasked to comply with the intent of RCRA to protect the environment and public health, while allowing for adequate public participation. Allowances for RD&D should also be made in other associated regulations.

The delisting change would minimize the expensive downtime of equipment and dedicated manpower at the demonstration site since the lengthy formal process involved with delisting would be completed prior to project start.

The alternatives discussed will significantly improve the timeliness of RD&D permitting under RCRA. Without these changes we will not have sufficient innovative, proven cost-effective technolology options to choose from when a ban on land disposal takes effect.

Literature Cited

1. Mills, E.S., The Economics of Environmental Quality, W.W. Norton & Company, Inc., New York, 1978.
2. Doull, J., Klaassen, C.D., Amdur, M.O., eds., Casarett and Doull's Toxicology. MacMillan Publishing Co., Inc., New York, 1980.
3. Goldstein, E.C., ed., Social Issues Resources Series, Pollution,

Vol. 3, Articles 1-100, 1980-1984, SIRS Inc., Boca Raton, Florida, 1985.

4. Arbuckle, J.G., Frick, F.W., Miller, M.L., Sullivan, T.F.P., and Vanderver, T.A., Jr., Environmental Law Handbook, Sixth Edition, Government Institutes, Inc., Washington D.C., 1979.
5. Memorandum from Marcia E. Williams, Director, Office of Solid Waste, to Hazardous Waste Division Directors, Hazardous Waste Branch Chiefs, Hazardous Waste Section Chiefs, Regions I-X, Oct 3, 1985, Subject: Research, Development, and Demonstration (RD&D) Permits.
6. Federal Register, Monday, January 14, 1985, Part II, Hazardous Waste Management System; Dioxin-Containing Wastes; Rule.
7. The Bureau of National Affairs, Environmental Reporter, August 22, 1986, EPA Grants Final Exclusion From RCRA for Wastes Generated at Eight Facilities, p. 611, BNA, Washington D.C., 1986.
8. U.S. EPA Office of Solid Waste, Petitions to Delist Hazardous Wastes - A Guidance Manual, EPA/530-SW-85-003, April 1985, NTIS Number PB85-194488.
9. U.S. EPA Office of Solid Waste, Guidance Manual for Research, Development, and Demonstration Permits Under 40 CRD Section 270.65, EPA/530-SW-86-008, July 1986, NTIS Number PB86-229192.
10. The Bureau of National Affairs, Environmental Reporter, February 7, 1986, Research, p. 1838, BNA, Washington D.C., 1986.
11. Hazardous Waste News, Vol. 8, No. 7, February 17, 1986, Business Publishers, Inc., Silver Spring MD, 1986.

RECEIVED November 25, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch017

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch018

Chapter 18

Risk-Qualified Mapping of Polychlorinated Dibenzodioxin Contamination

R. C. Bryan[1] and D. E. Splitstone[2]

[1]Geostat Systems, Inc., P.O. Box 1193, Golden, CO 80402
[2]International Technology Corporation, Regional Office, William Penn Plaza, 2790 Mosside Boulevard, Monroeville, PA 15146-2792

Nonparametric geostatistics is a useful tool for the adequate assessment of the magnitude and spatial distribution of alleged contamination at a potential hazardous waste site. The resulting mappings of expected contamination as well as risks of making incorrect remedial decisions can support the development of effective and cost-controlled site remediation. Because of the nature of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in soils, traditional geostatistical techniques provide little assistance in producing risk qualified mappings of site contamination. Nonparametric geostatistical techniques used to estimate the conditional probability distribution of an unknown concentration provide the means to produce such meanings. Using an "indicator" transformation of actual site TCDD data, these techniques are illustrated. This approach has several outstanding features:

- It is distributed free and resistant to outlier data; hence, it can be applied to data sets whatever the shape of the site data histogram.
- It results in confidence intervals which are not only data configuration dependent but also data values dependent.
- It is reasonably simple in its application and has performed well in the example site investigation.

An industrial site contaminated with polychlorinated dibenzodioxin (PCDD) presents a set of complex problems to those charged with the assessment of site contamination and the development of a remediation plan. In the suite of PCDD compounds, TCDD is of particular concern. The U.S. Environmental Protection Agency (U.S. EPA) has designated TCDD as a potent animal carcinogen and very acutely toxic(1).

0097-6156/87/0338-0246$06.00/0
© 1987 American Chemical Society

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch018

The first decision to be made from the results of a site assessment and subsequent risk analysis is whether to remediate a site or leave the contamination in situ. Frequently, site remedial alternatives may result in greater environmental risks than in situ containment. The evaluations of remedial alternatives often hinge on one's ability to accurately map the magnitude and extent of contamination at a site. Such mappings should also support an assessment of the risk or probability of not remediating a contaminated area of the site and the risk of unnecessarily remediating a noncontaminated area. The former probability may be associated with public health consequences; the latter can be linked to the unnecessary expenditure of remediation dollars. The application of geostatistical techniques to the mapping of site contamination can provide mappings which satisfy these desired objectives.

Zirshky, et al.(2), present an extensive review of classical geostatistical concepts as they apply to hazardous waste investigations. The basic concept of geostatistics is often referred to as the theory of regionalized variables. Briefly summarized, this theory states that observations which are close together are similar and the similarity between observations decreases as the distance between points of observation increases. The geostatistical measure of similarity among observations is one-half of the square of the difference between measurements taken a fixed distance apart. If the measure of similarity is averaged over all pairs of points a fixed distance apart and calculated over a range of distance separations between points, a graphical representation of the spatial similarity among observations can be constructed. This graphical representation is known as the variogram. An idealized variogram is presented in Figure 1.

Because some understanding of the information and geostatistical concepts presented by the variogram is required for an understanding of the application presented in this paper, a few of these concepts will be briefly presented. The idealized variogram presented in Figure 1 clearly illustrates the concept of spatial similarity mentioned above. As it is unlikely that observations taken close together will be exactly the same, the variogram model does not reach the distance axis (zero variation between points) at zero distance separation. This portion of the variation among observations is commonly referred to as the nugget and represents random variation which may be due in part to sampling and measurement variability. As the distance between observations becomes larger, the variogram approaches the "sill" which represents the total variability among the observations over the entire site. The distance at which the variogram reaches the sill is known as the "range."

Characterization of the variogram from actual observations permits the estimation of concentrations at points on the site which were not sampled by application of generalized least-squares type statistical regression algorithms. This type of estimation has come to be referred to as "kriging"(2). Thus, once the similarity of observations with distance has been described in terms of the variogram, contamination across the site can be estimated and mapped.

American Chemical Society
Library
1155 16th St., N.W.
Washington, D.C. 20036

For the typical site, characterization of the spatial similarity relationship among TCDD concentrations in soils in terms of the variogram is complicated by the very nature of TCDD. TCDD is tightly bound to soils containing a high organic content and is therefore not mobile in soil. Kearney, et al.(3), observed that the mobility of TCDD decreased with increasing organic content of soil. Similar conclusions were reached by Matsumura and Benezet(4) who showed that the mobility of TCDD is much slower than that of dichlorodiphenyltrichloroethane (DDT). Ward and Matsumura(5) observed that the concentrations of TCDD in waters separated from a contaminated lake bottom sediment exceeded its water solubility (0.2 parts per billion [ppb]). The report suggests this may be due to the binding or adsorption of TCDD onto organic matter or suspended sediment particles.

Jackson, et al.(6), examined the leaching potential of TCDD-contaminated soils from various sites. Soil partition coefficients were large (the mean log K_d for two samples was 5.9 and 6.02), indicating that TCDD is only very slightly soluble and that only very small amounts of TCDD will be released into water. The authors concluded that the rates of TCDD movement in soils are so slow that other transport mechanisms such as wind and water erosion are likely to be more significant.

Once TCDD reaches the soil, it will not spread in the absence of a solvent other than by some mechanical means. On an industrial site, the initial deposition of TCDD in soil quite likely resulted from accidental spills. Thus, it is easy to envision a situation where measurement of TCDD concentrations across the site reveals very few "high" concentrations and many concentrations are observed as "low" or not detected. Further, one can easily envision that both "high" and "low" measured concentrations might occur within a confined spatial volume of soil. Such is the case with the industrial site used as an example here.

The example site is located in the northeastern United States in an area that has been industrialized since the 1870s. The site occupies approximately four acres and is surrounded by other industrial facilities. Since the early 1900s, agricultural chemicals have been produced on the site by several manufacturers.

For a period of time in the 1950s and 1960s, these chemicals included 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). TCDD is a by-product of 2,4,5-T production and in reality is a product contaminant. Thus, it is plausible to consider TCDD being deposited at the site in the course of product spills and leaks. A major industrial accident occurred at the site some 20 years ago and subsequent reconstruction activities can be hypothesized to have a significant effect on the current deposition of TCDD at the site.

Samples of soil from the site indicated that TCDD concentrations of over 10,000 ppb were present. This discovery initiated further sampling to ascertain the extent and magnitude of contamination. Thirty near-surface (0 to 2 feet) borings were taken across the site to investigate the vertical and horizontal extent of TCDD contamination. Several deeper borings were also performed. The sampling program was designed to locate borings in areas suspected of contamination. Over 100 analyses for TCDD were performed which indicated that TCDD concentrations ranged from 0.02 to 19,500 ppb.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch018

The frequency distribution of the TCDD concentration data from the example site is presented in Figure 2. Note that the concentration scale in this figure is logarithmic, implying that a highly skewed frequency distribution is represented.

While the frequency distribution presented in Figure 2 might suggest that geostatistical analysis may be applied to the data after a logarithmic transformation, a closer examination of the data indicates that this is not the case. The observed concentrations for pairs of observations at the same depth and within as little as 60 feet of each other vary by orders of magnitude. The unhappy consequence of this is that the large variability among observations which are close together is reflected in the nugget of the variogram. This is true even if the logarithmic transformation is applied to the data. Thus, the geostatistical description of the similarity relationship among observations as a function of their distance apart would appear to be compromised. In addition, the use of "block kriging" to estimate the average concentration within a reasonable size remediation unit or "block" would appear to be inappropriate as the estimated "block" average would not reflect the physical reality unless the "block" size was chosen to be very small. Attempts at describing the within-block variability in terms of a convenient distributional assumption, say of a log-normal distribution, would also appear to be fruitless as the within-block variation is not proportional to the mean block concentration. Therefore, standard geostatistical techniques appear to be of little value in accomplishing the objective mapping of contamination in this situation.

Journel(7-9) provides a means for generating the desired mappings in situations such as the example site which are free from any undue assumptions regarding the distributional form of concentrations within blocks. This approach simply transforms the data into the sample cumulative probability distribution. This nonparametric geostatistical approach is sometimes called "indicator kriging."

The objective of indicator kriging is to estimate the local distribution of TCDD concentrations rather than the concentrations themselves. To apply indicator kriging, the data must be transformed from the original measurements of concentrations into indicator variables. This is accomplished by letting $z(x_a)$, a=1, N represent the initial data set

where

$z(x_a)$ = the TCDD concentration at datum location x_a, which is interpreted as a realization of the random variable, $Z(x_a)$

x_a = the location of the datum in space, given in three-dimensional coordinates, and

N = the total number of data.

The indicator transform of the datum, $z(x_a)$, is defined as:

$$i(x_a:z) = m \begin{matrix} 1, \text{ if } z(x_a) \text{ F } z \\ 0, \text{ if not} \end{matrix}$$

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch018

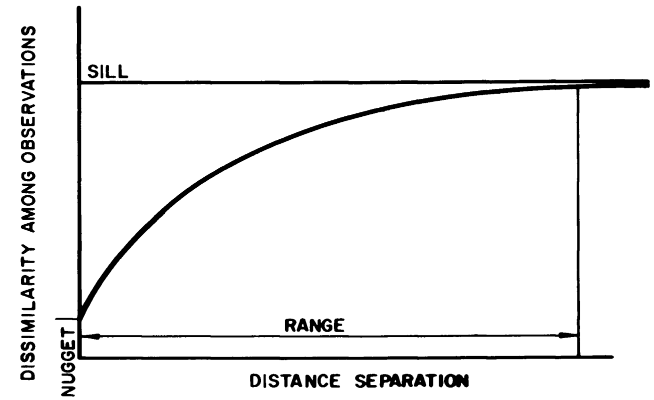


Figure 1. Idealized variogram.

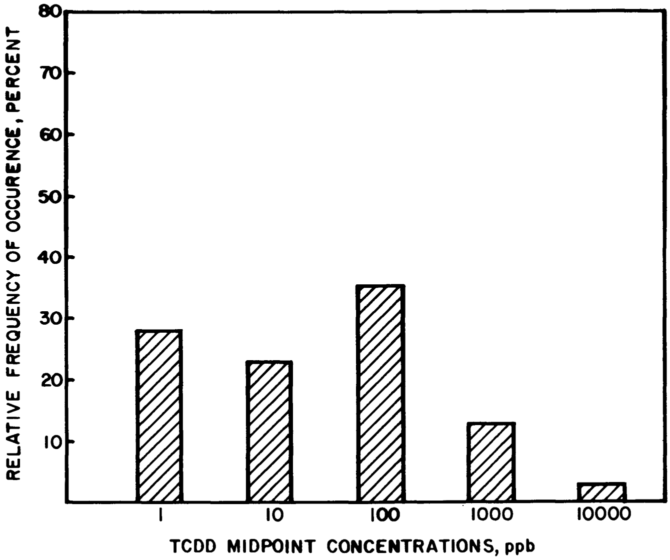


Figure 2. Histogram of TCDD concentrations at the example site.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch018

where

z = a "cutpoint" concentration.

The indicator transform was completed for six cutpoints. Table 1 presents these cutpoint concentrations, the number of observations between consecutive cutpoints, and the mean concentration for each class.

TABLE 1
INDICATOR CLASSES

CLASS	CUTPOINT CONCENTRATION	NUMBER OF OBSERVATIONS	CLASS MEAN CONCENTRATION (ppb)
1	-	56	9.8
2	50	26	135.3
3	275	13	398.6
4	675	7	874.1
5	1,700	3	2,240.0
6	2,700	2	2,715.0
7	3,500	4	8,937.5
		111	

The distribution of the random variable, $I(x_{az})$, associated with $i(x_a{:}z)$ is called a Bernoulli distribution, and its mean and variance are given by:

$$E[I(x{:}z)] = 1 \quad prob[z(x) \leq z] + 0 \quad prob[z(x) \geq z] = F(z)$$

$$Var[I(x{:}z)] = F(z) \quad [1 - F(z)]$$

where

$F(z) = prob[z(x) \leq z]$ is the stationary cumulative distribution function of the random variable $Z(x)$.

In practice, $F(z)$ is estimated by the cumulative histogram, $F^*[z(x_a)]$, of all the N data. If each datum receives the weight $1/N$, the cumulative histogram is given by:

$$F^*[z(x_a)] = (1/N) \quad t_a, \text{ for } a = 1, \ldots, N$$

where t_a is the rank order of the datum $z(x_a)$ which is an integer ranging from 1 (for the smallest datum) to N (for the largest datum).

The goal of indicator kriging is to estimate the conditional distribution function of the unknown concentrations, $Z(x)$ at location x, given "n" data within its neighborhood. Symbolically, this is represented as:

$$F_x[Z|\ (n)] = prob[Z(x) \leq z|\ (n)]$$

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch018

An estimate of the conditional distribution of Z(x) can be obtained by an ordinary kriging of the indicator transformed data.

The indicator kriging estimator, I*(x:z), appears as a linear combination of the indicator data, $i(x_a:z)$, in the neighborhood, "n," surrounding the point for which a prediction is to be made. This can be symbolized as:

$$I^*(x:z) = S_n \, w_a \;\; i(x_a:z)$$

where the weights, w_a, are derived from the variogram description of regional continuity. I*(x:z) is an estimate of the conditional distribution function of Z(x) given the data. Note that there are as many kriging estimators as there are cutpoints. In the present case, there are six such estimators.

Variogram Description of Continuity

The spatial continuity relationship (correlation) for each indicator, $I(x:z_k)$, was characterized by an empirical variogram model of the apparent continuity within the near-surface soil in each of two dimensions. The construction of such a model is based on the assumption that the value of the indicator at location x, i.e., $I(x:z_k)$, may be greatly influenced by values of that indicator at nearby locations and less influenced by more distant values. This degree of relationship between points a specified distance, h, apart may be measured by:

$$q(h) = 1/2 \; [I(x:z_k) - I(x+h:z_k)]^2$$

Averaging all differences between pairs of points separated by a distance, h, gives rise to the variogram.

The function q(h) is directly related to the coefficient of correlation, r(h), between two indicator values a distance h apart by:

$$r(h) = 1 - \frac{q(h)}{Var}$$

where Var represents the total variance among the indicator values.

The experimental variogram models constructed for each of the six cutpoints indicated that a simple omnidirectional variogram model was an appropriate descriptor of the continuity relationship for each of the six cutpoints. Figure 3 presents the variogram model appropriate for the 50 ppb cutpoint.

This variogram shows a nugget (Co) of 0.12 with a sill of 0.253. The indicator variogram reaches the sill at a range, A, of 90 feet.

Indicator Kriging

The characterization of the spatial distribution of TCDD concentrations was performed by obtaining estimates of the conditional probability distribution function, $I^*(x:z_k)$, for each of the six concentration cutpoints, z_k, k=1,. . .,6. These estimates

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch018

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch018

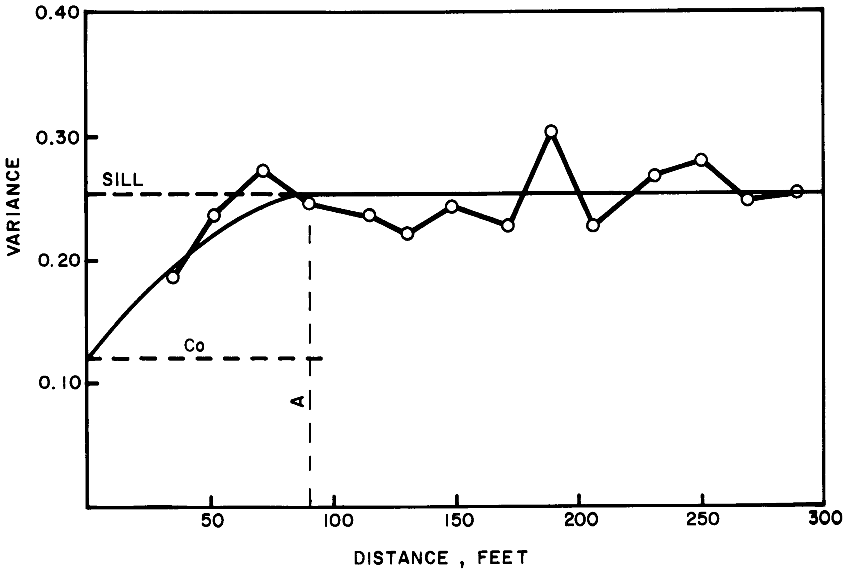


Figure 3. Indicator variogram appropriate for the 50-ppb cutpoint.

of the probability that a cutpoint concentration will not be exceeded were obtained at each node of a 40-by-40-foot grid system superimposed on the site.

In practice, the conditional probability distribution function, $F_x[z|(n)]$, is estimated by a discrete set of k values, $I^*(x:z_k)$, one of each of the k cutpoints. In this case there are six such values. Since each estimate is determined individually, there is no guarantee that $I^*(x:z_k)$ will verify the order-relations of a cumulative distribution function, i.e.:

$$I^*(x:z_{k-1}) \ F \ I^*(x:z_k) \quad \text{for } k=2,\ldots,6$$

In cases where order relation problems presented themselves, correction estimates were obtained using a simple averaging of the points in the region of the problem. This is consistent with the procedure recommended by Isaaks(10) and the results are nearly identical to more sophisticated averaging algorithms. Once these ordering problems are resolved, several types of data presentations are possible.

Data Presentations

The conditional probability of an unknown TCDD concentration in an area being less than a threshold, T, can be found by simple linear interpolation from the estimated conditional probability distribution function. This interpolation is of the form:

$$\text{prob}[Z(x) \ F \ T|(n)] = I^*[x:z_{k=1}|(n)] +$$
$$[I^*[x:z_k|(n)] - I^*[x:z_{k-1}|(n) \quad (T-z_{k-1})/(z_k-z_{k-1})$$

The conditional probability of an unknown TCDD concentration being greater than the threshold, T, is just one minus the probability given by the previous equation, i.e.:

$$\text{prob}[Z(x) > T|(n)] = 1.0 - \text{prob}[Z(x) \ F \ T|(n)]$$

The isopleths of the probability that an unknown TCDD concentration exceeds 50 ppb and 675 ppb are presented in Figures 4 and 5, respectively.

The expected value of an unknown concentration of TCDD, Z(x), can be estimated using the following approximation to its conditional expectation:

$$E^*[Z(x)|(n)] = \overset{k+1}{\underset{1}{S}} u_k[I^*[x:z_k|(n)] - I^*[x:z_{k-1}|(n)]$$

where:

$$I^*[x:z_{K+1}|(n)] = 1.0$$

$$I^*[x:z_0|(n)] = 0.0$$

and

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch018

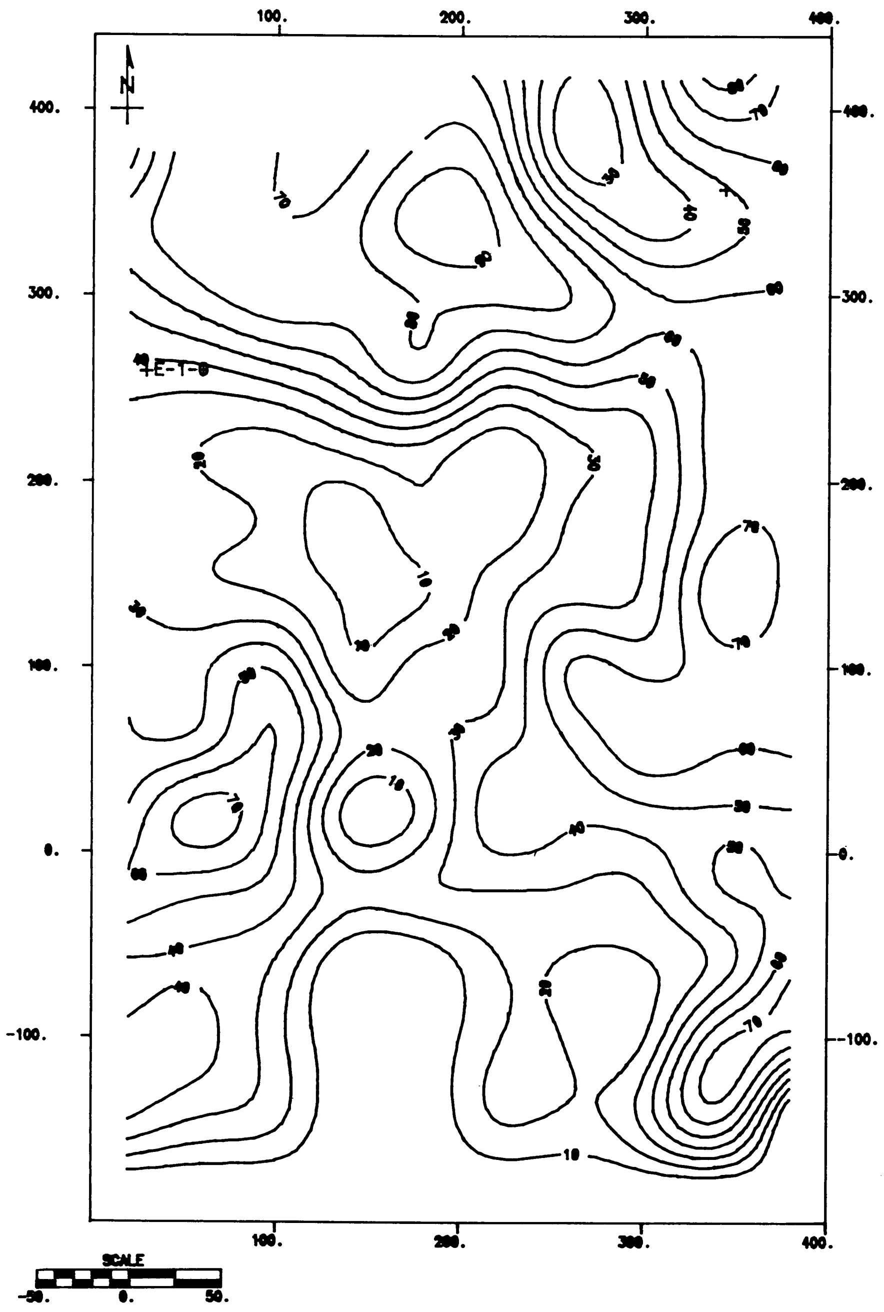


Figure 4. Isopleths of probability that an unknown TCDD concentration exceeds 50 ppb.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch018

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch018

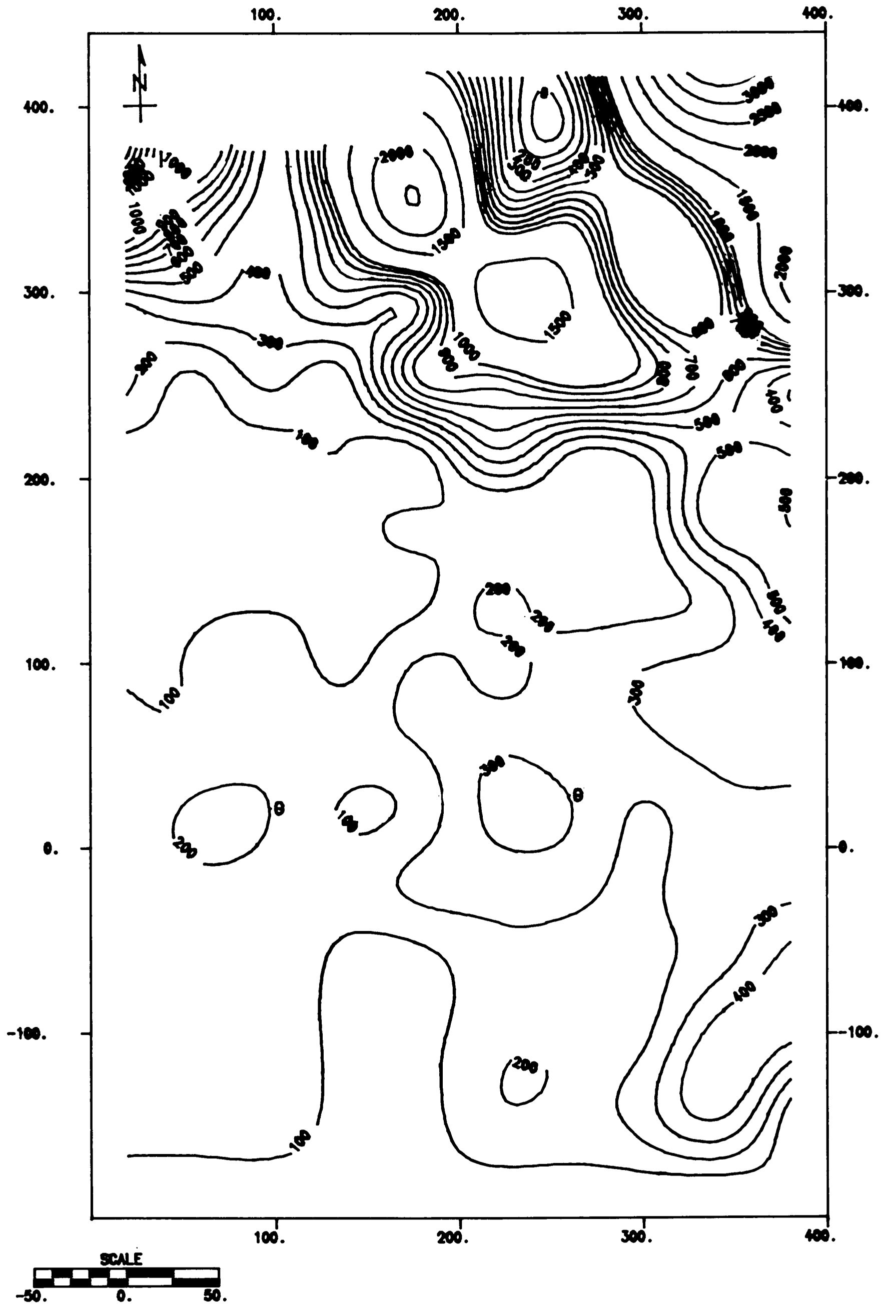


Figure 5. Isopleths of probability that an unknown TCDD concentration exceeds 675 ppb.

In Solving Hazardous Waste Problems; Exner, J.;
ACS Symposium Series; American Chemical Society: Washington, DC, 1987.

u_k = the mean concentration of the class of data between cutpoint concentrations z_k and z_{k-1}. The class mean concentrations for the seven classes are given in Table 1.

For instance, at the point 180 East, 380 North, the probability that the TCDD level is below the concentration cutpoint of 50 ppb is 0.21 and the probability that the TCDD level is below the concentration cutpoint of 2,700 ppb is 0.89. After generating the probability estimate for each cutpoint at this location, the conditional expectation for the TCDD concentration can be calculated as 882.42 ppb. Table 2 illustrates this calculation.

Performing similar calculations for all points on the 40-by-40-foot grid permits the mapping of the expected values of TCDD concentrations across the site. Such a map is presented in Figure 6.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch018

TABLE 2

PROBABILITIES AND ESTIMATE OF TCDD AT 180 EAST, 380 NORTH

THRESHOLD	CUTOFF CONCENTRATION CLASS	CONDITIONAL PROBABILITY	PROBABILITY WITHIN CLASS	CLASS MEAN CONCENTRATION	AVERAGE PROBABILITY TIMES CONCENTRATION
	0 ppb				
			0.21	9.8	2.06
1	50 ppb	0.21			
			0.21	135.3	28.41
2	275 ppb	0.42			
			0.16	398.6	63.78
3	675 ppb	0.58			
			0.15	874.1	131.12
4	1,700 ppb	0.73			
			0.16	2,240.0	358.40
5	2,700 ppb	0.89			
			0.11	2,715.0	298.65
6	3,500 ppb	1.00			
			0.00	8,937.5	0.00
	99,999 ppb	1.00			
TOTAL			1.00		882.42

Conclusions

The indicator approach to estimation of spatial distributions of TCDD concentrations consists in estimating first the conditional probability distribution of any unknown concentration. Estimates of the unknown concentration are then derived, together with their confidence intervals. This approach has several outstanding features:

- o It is distribution free and resistant to outlier data; hence, it can be applied to data sets whatever the shape of the site data histogram.
- o It results in confidence intervals which are not only data configuration dependent but also data values dependent.
- o It is reasonably simple in its application and has performed well in the example site investigation.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch018

Literature Cited

1. U.S. Environmental Protection Agency (U.S. EPA), 1984, "Health Assessment Document for Polychlorinated Dibenzo-p-Dioxins," EPA 600/8-84-014A.
2. Zirshky, J., G. P. Kary, R. O. Gilbert, and J. E. Middlebrooks, 1985, "Spatial Estimation of Hazardous Waste Site Data," Journal of Environmental Engineering, Vol. III, No. 6, December 1985, pp. 777-789.
3. Kearney, P. C., A. R. Isensee, C. S. Helling, E. A. Woolser, and J. R. Plimmer, 1973, "Environmental Significance of Chlorodioxins," Advances in Chemistry, Series 120, pp. 105-111.
4. Matsumura, F. and H. J. Benezet, 1973, "Studies on the Bioaccumulation and Microbial Degradation of 2,3,7,8-Tetrachlorodibenzo-p-dioxin," Environmental Health Perspectives, Vol. 5, pp. 253-258.
5. Ward, C. and F. Matsumura, 1978, "Fate of 2,3,7,8-TCDD in a Model Aquatic Environment," Archives of Environmental Contamination and Toxicology 7 (3) pp. 349-357.
6. Jackson, D. R., M. H. Roulier, H. M. Grotta, S.W. Rust, J. S. Warner, M. F. Arthur, and F. L. DeRoos, 1985, "Leaching Potential of 2,3,7,8-TCDD in Contaminated Soils," Proceedings of the Eleventh Annual Research Symposium, EPA-600/9-85-013, pp. 153-168.
7. Journel, A. G., Non-Parametric Estimation of Spatial Distribution, Math. Geol., 1983, 9, No. 6, pp. 563-586.
8. Journel, A. G., New Ways of Assessing Spatial Distributions of Pollutants, in Environmental Sampling for Hazardous Wastes, G. Schweitzer, Ed., Amer. Chem. Soc. Press, 1984, pp. 109-118.
9. Journel, A. G., Indicator Approach to Toxic Chemical Sites, EPA Project Report No. CR-811235-02-0, EPA Exposure Assessment Division EMSL, Las Vegas, 30 p., 1984.
10. Isaaks, E. H., Risk Qualified Mappings for Hazardous Waste Sites. A case study in distribution-free geostatistics, MSc. thesis, Applied Earth Sciences Department, Stanford University, 1984.

RECEIVED December 2, 1986

Chapter 19

Analytical Support During Remedial Action at Sites Contaminated with 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin

Robert Kleopfer, Mary Gerken, Angelo Carasea, and Debra Morey

U.S. Environmental Protection Agency, 25 Funston Road, Kansas City, KS 66115

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch019

The U.S. Environmental Proection Agency (EPA) has identified more than 40 sites in Missouri which are contaminated with 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) at levels above one part per billion (micrograms per kilogram) in soil. Remediation at some of these sites began in 1985, with excavation and temporary storage of the contaminated soil. Timely analytical support was needed to optimize the utilization of field resources while monitoring for potential exposure to local residents. Standard analytical procedures used during the preliminary site investigations were not suitable for the remedial phase due to unacceptable turnaround time in reporting validated data. Analytical procedures were consequently modified to reduce data turnaround time. Procedures were also developed for semi-automated data validation and electronic data transmission from the analytical laboratories to EPA.

The U.S. Environmental Protection Agency has identified more than 40 sites in Missouri which are contaminated with 2,3,7,8-TCDD at levels above one part per billion (micrograms per kilogram) in soil. In each case, the contaminant originated from the manufacture of hexachlorophene at a chemical plant in Southwestern Missouri (1). In most instances, the contamination occurred during the application of waste oil for the purpose of dust control. Sites include an entire town (Time Beach), a mobile home park (Quail Run), residential streets, rural roads, horse arenas, small farms, streams (Romaine Creek and Spring River), and parking lots. Although most of the contamination is limited to soil, 2,3,7,8-TCDD has also been found in house dust, mobile home insulation material, concrete slabs, tree bark, stream sediment, and biota (worms, fish, etc.).

This chapter not subject to U.S. copyright.
Published 1987, American Chemical Society

The public health implications required a rapid, yet reliable assessment of these sites. A cost-effective procedure was developed to analyze for 2,3,7,8-TCDD down to 1 part per billion (ppb) in hundreds of soil samples (2). From late 1982 thru 1985, over 13500 samples were analyzed by 50 different laboratories using procedures based on isotope dilution and high resolution gas chromatography with low resolution mass spectrometry. A comprehensive quality assurance program was implemented to assure the reliability of data generated during this massive monitoring effort (3).

Remediation Options

A number of different options have been proposed for remediation of the Missouri sites (4). The two basic proposals are removal and in-place treatment. Post-removal options include:
1. Immediate treatment or detoxication of the excavated soil,
2. Temporary storage of all contaminated soils, and
3. Long-term storage in an approved landfill, concrete bunker, or abandoned mine. In-place treatment proposals have included biodegradation, chemical degradation, photolysis, and chemical stabilization.

In 1980, Syntex, Inc., utilized a process based on photolysis to destroy approximately 13 pounds of 2,3,7,8-TCDD contained in 4300 gallons of oily trichlorophenol still-bottom residues (5). In 1984, EPA began to use a transportable rotary kiln incinerator to detoxify solid and liquid waste which were contaminated with 2,3,7,8-TCDD (6). Concurrent with the incineration activities, EPA began removing soils and other contaminated materials from a number of sites including a mobile home park near Grays Summit, Missouri. These activities required the application of extensive compositing techniques and statistical treatment of the data to assure the removal of 2,3,7,8-TCDD down to acceptable levels (7).

Analytical Requirements

During remedial operations, large costs can be incurred if cleanup operations are delayed by slow response in the analysis of samples. Thus, analytical techniques must be available to verify that a cleanup criterion has been achieved and to indicate during cleanup efforts whether to stop or proceed. Preliminary discussions of proposed excavation approaches led to the requirement of 20 to 25 analyses within a 24-hour period. The standard EPA contract laboratory procedure required a minimum of 72 hours elapsed time including data reporting and data validation by EPA. Unfortunately, available protocols such as the EPA Contract Laboratory (CLP) mechanism, typically required two weeks or longer to produce verified data. Alternatives were needed to meet the unique demands of site remediation.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch019

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch019

EPA Contract Laboratory Procedure

This procedure (2) has been used since 1982 by EPA for routine investigations. The analytical protocol requires complete extraction of the sample followed by liquid chromatographic cleanup and isomer-specific 2,3,7,8-TCDD analysis by high resolution gas chromatography/low resolution mass spectrometry. The extraction begins with ten grams of soil which is spiked with internal ($^{13}C_{12}$-2,3,7,8-TCDD) and surrogate ($^{35}Cl_4$-2378-TCDD) standards. A drying agent (anhydrous sodium sulfate) is added and the mixture is allowed to stand for a total of six hours. After this period, 170 ml of (methanol/hexane) is added to the sample and the mixture is shaken for at least three hours. The sample is filtered and the jar and soil are thoroughly rinsed with additional solvent. The combined rinses and original extracts are concentrated to about 1 ml by Kuderna-Danish evaporation. The concentrated extract is then cleaned up by elution in series through a neutral/ acidic/basic silica gel column and an alumina column. The eluted fraction containing dioxin is collected and concentrated. Cleanup by carbon column chromatography is required as a final polishing step. Further separation of the sample components uses a fused-silica capillary column (SP-2340 or equivalent) before entering the mass spectrometer for analysis. Data repdrts are prepared and shipped (generally by air express) to EPA for review and data validation. The elapsed time requirements for these processes are summarized in Table I.

Modified LRMS Procedure

The previously described contract laboratory procedure was modified to permit faster data turnaround. The major changes included the following:

1. The six hour drying time requirement was deleted. Anhydrous sodium sulfate was still required however.
2. The extraction solvent was changed from 12% methanol/hexane to 10% acetone/hexane and the extraction time was reduced from three hours to one hour. A sonication step was added, however.
3. A three concentration level (1, 5, and 25 ppb) calibration curve is required rather than five.
4. The requirement to verify isomer specificity was deleted since TCDD isomer interference was not a problem at the Missouri sites (1).

Tandem Mass Spectrometry Procedure

The application of GC/MS/MS to the analysis of 2,3,7,8-TCDD in soil was first demonstrated in 1983 (8) and later refined (9,10,11).

Five grams of anhydrous sodium sulfate is placed in a 10 ml serum vial and the vial, with cap and septum, is weighed. Approximately five gram of a soil sample is added and the vial is reweighed. The sample is spiked with internal and surrogate

standards of isotopically labeled 2,3,7,8-TCDD. The sample is mixed by shaking and extracted with 5 ml of 67% acetonitrile/dichloromethane in the closed vial. An aliquot of the extract is taken and, after separation from acetonitrile, the dichloromethane is used directly for GC/MS/MS analysis. Cleanup should usually not be necessary, but a cleanup procedure is included for those samples which do not meet quality assurance criteria. A short capillary column (15m DB-5) is used which allows for separation of TCDD from the bulk sample matrix and measurement of TCDD in the extract. Quantification is based on the response of native TCDD relative to the isotopically labeled TCDD internal standard.

The method employs a tandem quadrupole mass spectrometer (MS/MS) as the final detector. The specificity of detection inherent in such a system significantly reduces the need for sample cleanup. This, in turn, improves productivity and cost-effectiveness relative to other high resolution and low resolution GC/MS analysis techniques. The aparatus and methods described are designed for use in a mobile laboratory, which permits on-site analyses.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch019

Data Handling/Data Review

The Contract Laboratory Program procedure required delivery of specified documents included data summary forms, calibration data, chromatograms, and mass spectra and were generally shipped by air freight. Upon receipt by EPA, the documents were reviewed for data usability which was based on consideration of completeness of documentation and adherence to quality control requirements (3). Due to the nature of the remedial projects, these procedures had to be modified to allow for quicker turnaround.

To achieve this quicker turnaround, EPA Region VII needed a method that not only allowed for the quick transfer of raw data, but a system to quickly review, validate, and transmit the data. To support this goal, a computer system was developed which allowed for the circular transfer of information via computers from the EPA field team to the lab; the raw data from the lab to EPA; and validated results from EPA back to the field team.

The laboratory portion of the computer system was made up of a series of computer programs written in dBASE III. These programs were designed to work on any IBM portable, XT, or AT computer or compatible models. The actual data software is composed of three to four inter-related dBASE files (labs using GC/MS require three files while labs using GC/MS/MS require four files) linked together through the use of a common file name. A different extention is given to each file name for distinction among the files for a particular set of samples.

Each of the inter-related dBASE files allows for specific data entry for a particular aspect of the sample set. Data are entered into individual files for the Initial Calibration; Blank Response (GC/MS/MS only); Continuing Calibration; and

actual sample results. Raw data entries are made into the respective files using screen driven monitor prompts. The software programs then compute all calculations required by the method. For example, initial calibration data are entered into the Initial Calibration file where it is used to calculate the native TCDD and surrogate RRFs; ion ratios; mean RRFs; %RSDs; and standard deviations. The computer program then compares these results to established criteria. Any data not meeting the criteria are flagged. Each of the other files work in the same manner by flagging unacceptable data. Not only does this flagging of data assist the EPA data reviewer in spotting unacceptable data, it gives the lab an opportunity to rerun any samples not meeting criteria prior to turning it in.

The inter-related dBASE files representing the data set are transmitted to EPA via Crosstalk software over regular commercial phone lines. The EPA data reviewer will print out the data files looking for unacceptable data or anomalies which may be present. Since the computer program has done all the data calculations, these will not be verified by the data reviewer, saving much time. Data are reviewed much the same way as for routine work with the exception that only the computer transmitted raw data will be used for the initial qualification of the data. The actual mass chromatograms and spectra are received seven days later at which time they are also reviewed. Any discrepancies between the two sets of data are immediately rectified. Data are qualified based on adherence to the method criteria plus the results of the field blank, fortified sample, and performance evaluation sample. Data are qualified as either valid or invalid for remedial site work.

The computer programs allow for the qualification of data and other data editing by EPA. The qualified data results are then fed into a dBASE text file which forms a memorandum which will be used to formally transmit the data to the field team. This dBASE text file containing the data transmittal memorandum is sent electronically to the field team.

Method Performance

Before implementation of the proposed procedures, a round-robin comparison study was performed which involved three different soils, three different methods, and five different laboratories. These results are summarized in Table II. Although the modified procedures gave somewhat lower mean results, the differences were not statistically significant. The MS/MS procedure displayed similar performance characteristics during its utilization in 1985 at a remedial site in Eastern Missouri. Those results are summarized in Table III.

Conclusions

By modifying analytical procedures and relying on modern PC based data management and communications technology, we have succeeded in providing timely analytical data to support hazardous waste

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch019

site cleanup activities. The system described was used to assist in the cleanup of one mobile home park in Eastern Missouri over a two year period. Numerous other applications have been started or are planned.

Table I. Analytical Turn Around Times For 2,3,7,8-TCDD Determinations*

STEP	CLP	MODIFIED CLP	MS/MS
	PROCEDURE		
SAMPLE EXTRACTION[a]	12	3	1
CLEANUP OF EXTRACT[b]	10	4	3
GC/MS ANALYSIS[c]	12	12	6
DATA REDUCTION/TRANSMISSION[d]	25+	1	1
DATA VALIDATION[e]	4	1	1
TOTALS[f]	63+	21	12

*Estimated elasped times in hours for processing, analyzing, and reporting one batch of 24 soil samples.
a. Assumes two workers. Includes all weighing, drying, and extraction steps.
b. Assumes two workers. Includes all column chromatography and extract concentration steps.
c. Assumes that GC/MS/DS instrument has been previously calibrated. Assumes one instrument only.
d. Requires electronic or hard copy delivery of data in EPA specified format.
e. Includes data review and EPA decision on data usability.
f. The totals assume that each step is done sequentially. In practice, the processing steps may overlap thus reducing the total turn around times.

Table II. Methodology Comparison Data For 2,3,7,8-TCDD Contaminated Soils[a]

METHOD (LAB)	SOIL A		SOIL B		SOIL C	
	MEAN	RSD	MEAN	RSD	MEAN	RSD
CLP (1)	.81	20%	16.6	6%	<.053	N.A.
CLP (2)	.75	29%	15.8	13%	<.024	N.A.
CLP (3)	.83	11%	----		----	
RAPID LRMS(1)	.63	8%	15.5	6%	<.035	N.A.
RAPID LRMS(2)	.57	12%	13.6	4%	<.035	N.A.
RAPID LRMS(3)	.53	23%	13.4	13%	<.14	N.A.
MS/MS (4)	.51	27%	14.1	14%	<.3	N.A.
MS/MS (5)	.88	40%	15.7	10%	<.04	N.A.

a. Data are in micrograms per kilograms (ppb) based on six replicate runs by each laboratory and each soil. The Missouri soils were dry and well-homogenized.
RSD = Relative Standard Deviation
N.A. = Not applicable

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch019

Table III. Summary Of QC Results For Tandem Mass Spectrometry Procedure[a]

QC TYPE	MEAN	RSD	N
Soil A	.59	24%	24
Soil B	13.5	6%	26
Soil D	.91	20%	22
Soil Spike[b]	1.02	7%	64
Soil Blank[c]	<.03	NA	64

a. Data generated during 1985 remedial support.
b. Clean soil spiked at 1.0 PPB by laboratory.
c. Soil provided by EPA known to be uncontaminated by 2,3,7,8-TCDD.
N = Number of samples analyzed

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch019

References

1. R.D. Kleopfer, Chemosphere 14, 739 (1985).
2. R.D. Kleopfer, K. Yue, and W.W. Bunn, Chapter 27 in Chlorinated Dioxins and Dibenzofurans in the Total Environment-Volume II, L. H. Keith, C. Rappe, and G. Choudhary, Eds. (Stoneham, MA: Butterworth Publishers, 1985).
3. R.D. Kleopfer and C. Kirchmer, Chapter 26 in Chlorinated Dioxins and Dibenzofurans in the Total Environment-Volume II, L. H. Keith, C. Rappe, and G. Choudhary, Eds. (Stoneham, MA: Butterworth Publishers, 1985).
4. "Final Report of the Missouri Dioxin Task Force," submitted to Governor Christopher S. Bond (State Capitol, Jefferson City, MO 65101) October 31, 1983.
5. Exner, J. H., J. D. Johnson, O. D. Ivins, M. N. Wass, and R. A. Miller, Chapter 17, in Detoxication of Hazardous Waste (Ann Arbor, Michigan: Ann Arbor Science Publishers, 1982).
6. R. D. Kleopfer, F. J. Freestone, R. Hazel, and P. desRosiers, "Destruction of Dioxin Containing Wastes In a Mobile Incineration System," Chapter 34 in Chlorinated Dioxins and Dibenzofurans in Perspective, C. Rappe, G. Choudhary, and L. H. Keith, Eds (Chelsa, MI: Lewis Publishers, Inc., 1986).
7. J. H. Exner, W. J. Keffer, R. O. Gilbert, and R. R. Kinnison, "A Sampling Strategy for Remedial Action at Hazardous Waste Sites: Cleanup of Soil Contaminated by Tetrachlorodibenzo-p-dioxins," Chapter 10 in Chlorinated Dioxins and Dibenzofurans in Perspective" C. Rappe, G. Choudhary, and L. H. Keith, Eds (Chelsa, MI: Lewis Publishers, Inc., 1986).
8. Unpublished Report by Roy F. Weston, Inc., West Chester, PA. (1983).
9. "The Application of Flash Chromatography-Tadem Mass Spectrometry to the Analysis of Complex Samples for Chlorinated Dioxins and Dibenzofuran," T. Sakuma, N. Gurprasad, S. D. Tanner, A. Ngo, W. R. Davidson, H. A. McLeod, and B. Lau, Chapter 12 in Chlorinated Dioxins and Dibenzofurans in the Total Environment-Volume II, Chapter 12 (1985). L. H. Keith, C. Rappe, and G. Choudhary, Eds. (Stoneham, MA: Butterworth Publishers, 1985).

10. B. Shushan and S. D. Tanner, Chemosphere 14, 843 (1985).
11. "Comparison of a New Rapid Extraction GC/MS/MS and the Contract Laboratory Program GC/MS Methodologies, for the Analysis of 2,3,7,8-TCDD," J. S. Smith, D. Ben-Hur, M. J. Urban, R. D. Kleopfer, C. J. Kirchmer, W. A. Smith and T. S. Viswanethan, Chapter 25 in Chlorinated Dioxins and Dibenzofurans in Perspective, C. Rappe, G. Choudhary, and L. H. Keith, Eds. (Chelsa, MI: Lewis Publishers, Inc., 1986).

RECEIVED November 25, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch019

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch020

Chapter 20

Ambient Air Monitoring During the Cleanup of Hazardous Waste Sites Contaminated by Polychlorinated Dioxins

Tony Babb

International Technology Corporation, Air Quality Services Group, 3028 East Magnolia Avenue, Knoxville, TN 37914

Ambient air monitoring during the cleanup of hazardous waste sites contaminated by polychlorinated dioxins is done because of potential hazards associated with dioxin exposure. An effective ambient air monitoring program can document the exposure levels on site and the potential migration of contamination off site. The method validation data described herein provides a basis for concluding that the PUF high-volume sampler effectively collects dioxin with no observed breakthrough in a 24-hour sampling period. The PUF sampler with its first-stage particulate filter allows for quick, low cost determination of suspended particulate concentrations. This data, in many cases, may enable screening of samples for dioxin analysis based on the assumption that the higher the suspended particulate matter concentration at the site, the higher the potential for measurable levels of dioxin. Much more data is needed to further evaluate the effectiveness of the PUF sampler for measuring ambient levels of dioxin and to evaluate sample screening techniques which can be used to provide high quality data at a much lower cost.

For some animal species 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD, dioxin) is one of the most toxic synthetic compounds known to man (1). In addition to being toxic, 2,3,7,8-TCDD is also a suspected carcinogen. Dioxins can enter the environment in a variety of ways, generally as a contaminant of a product, as a waste by-product, or as a product of combustion. For these rea-

0097-6156/87/0338-0267$06.00/0
© 1987 American Chemical Society

sons, there is an immediate concern of the contamination of our environment with dioxins. Remedial action is ongoing to remove dioxin contaminated soil and debris from identified sites to safe storage until procedures to detoxify the material are available.

Due to the hazards associated with dioxin exposure, remedial action at dioxin contaminated sites presents a potential hazard to the general populace in the vicinity of the sites and to the personnel actually performing the remedial activity. As an issue of major concern to the public, regulators, industry, and scientists, the need to evaluate and control this potential exposure during remediation may be as important as the remedial action itself. An effective ambient air monitoring program can provide documentation of the exposure levels on site and of the potential migration of contamination off site. It can be used to assess the effectiveness of dust suppression techniques being used on site. It can identify and document meteorological conditions which may represent high potential for exposure. This paper summarizes information which has been used in the development and operation of several ambient air monitoring programs during the cleanup of sites contaminated with dioxin.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch020

Ambient Air Monitoring Survey Design

The proper development of a cost-effective air monitoring survey involves not only air quality monitoring but also meteorological monitoring, quality control, quality assurance, and data evaluation. Failure to recognize this fact at the outset results in a design based on many practical compromises that may fail to meet the sampling objectives. A cost-effective system should reflect both the realities of current air quality monitoring system technology and the ultimate application for which the measurement system is intended. It is especially important to develop a systematic plan for the implementation of the system in advance of choosing specific pieces of hardware (2).

Before developing a working air monitoring survey, specific general information about the contaminated site is needed. 1) What is there? It is important to know all the pollutants present because of the possibility of additive health effects and the possibility of tracking several parameters by monitoring only a few because of their chemical and/or physical characteristics. 2) How much is there? The answer to this question will aid in the determination of the type of monitoring equipment needed in terms of detection limits, averaging times, and estimating potential emission rates. 3) Where is the contamination? 4) What are the prevailing meteorological conditions? 5) Where are the receptor locations? This information will help to determine the physical size of the air monitoring survey, the number of air monitoring stations, the monitoring station locations and the duration of the survey.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch020

There are seven basic steps in the design of an air monitoring survey:

(1) Clearly define the objectives,
(2) Determine the parameters to be measured,
(3) Site selection,
(4) Determine sampling schedules and sampling duration,
(5) Determine sampling and analytical methods,
(6) Develop a quality control and quality assurance program, and
(7) Data management and reporting.

Objectives

The starting point is the setting of objectives. When monitoring during the cleanup of sites contaminated by dioxin, these objectives may include the following:

(a) Evaluating the potential for airborne migration of dioxin contamination off-site (general population exposure).
(b) Evaluating the potential for worker exposure on-site during remedial activities.
(c) Evaluating the potential for recontamination of areas already "cleaned".
(d) Evaluating the adequacy of methods being employed to contain contamination on site as a function of site activity and site-specific meteorological conditions.

Translating the study objectives into clear meaningful statements will serve as the cornerstone for the rest of the air monitoring plan.

Parameters To Be Measured

Once the objectives are clearly defined, the second step is to determine the physical parameters to be measured. In order to achieve any one or a combination of the previously listed objectives, it is necessary to measure ambient air concentrations of dioxin. In order to interpret variations in the distribution of air pollution concentrations, the on-site measurement of meteorology and a basic understanding of the role of the atmosphere in transporting and dispersing air pollutants is needed.

Sampling Site Selection

Step three is the selection of sampling sites. In choosing a technically suitable location for an air quality monitoring site, one must consider the representativeness of the site in terms of its exposure to air pollutants and prevailing meteorological conditions. Site exposure is heavily dependent on the relative location of pollutant sources and the effects of terrain on meteorological conditions. Where the maximum background concen-

trations are expected to occur and where the maximum project contribution to air quality is expected to occur should be considered when locating specific sites. The location of human receptors should be taken into account. It should be recognized that site selection is a critical element in the survey design. If the wrong site is picked or if a critical site is missed, no amount of accurate data collection will allow the objectives of the study to be fully realized.

The meteorological exposure of an air monitoring site should be representative of either area-wide meteorology or localized meteorological regimes that will affect the dispersion of air pollutants. Localized meteorological regimes result from irregular terrain features which can cause increased mecanical turbulence or localized drainage winds.

The number of monitoring sites that should be used in the study depends on the diversity of the land use patterns, meteorological regimes, source configurations, and sensitive receptors. This number is limited by the number of technically suitable sites available, the constraints of money and manpower available, and the level of statistical significance or confidence desired in the results.

Sampling Schedules and Sampling Duration

The fourth step is to determine the sampling schedules and duration. Due to the concern about hazards associated with dioxin exposure, the sampling schedule and duration should be continuous during the period in which there is remedial activity on site. This practice will probably continue until information pertaining to the hazards of exposure to dioxin indicate it is not necessary.

Sampling and Analytical Methods

The fifth step in the design of this air monitoring survey is determining the sampling and analytical methods to be used. The criterion generally used as an acceptable level of exposure to dioxin is in the very low picogram per cubic meter range. In order to monitor for the concentration of dioxin in ambient air at these levels, it is necessary to obtain sufficient sample volumes. The Environmental Protection Agency has developed a high volume sampler, referred to as a PUF Sampler, which through testing and actual field use appears to be suitable for this task (3) (4). Figures 1 and 2 show diagrams of the sampler and sampling module, respectively. The sampler utilizes a dual-chambered aluminum sampling module which contains both particulate and vapor phase collection media. The upper chamber supports a four-inch diameter airborne particulate glass-fiber filter. The lower chamber houses a glass cartridge which contains the polyurethane foam plug for vapor entrapment. This glass cartridge may readily be used with several different types of solid sorbents. Air flow is provided by a high pressure blower. Air flow rate is monitored with a

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch020

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch020

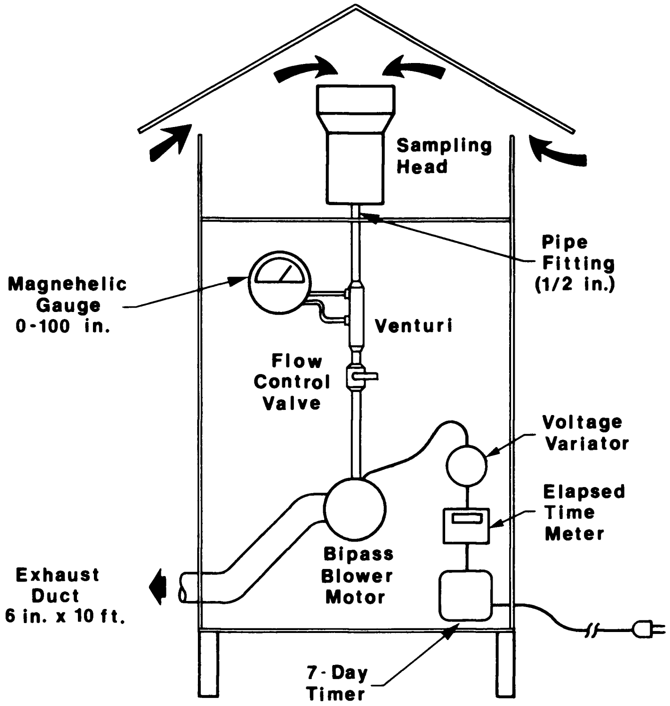


Figure 1. Modified Hi-Vol Sampler (General Metal Works, Inc., Model PS-1) Used To Collect Suspended Particulate Matter (PM) and 2,3,7,8-TCDD Samples

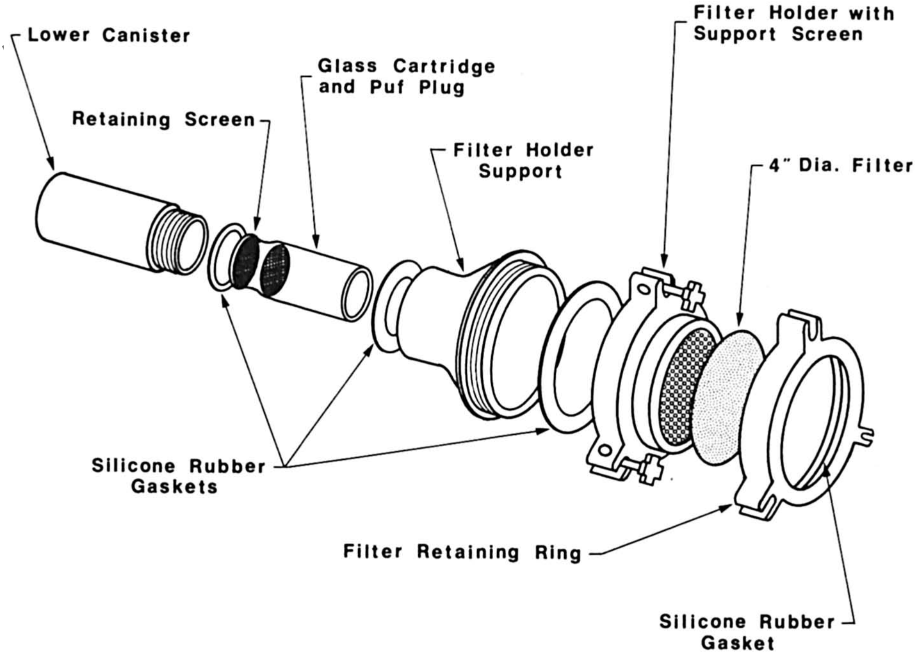


Figure 2. A Detailed Illustration Of The Sampling Head Module Showing The Fiberglass Filter Used To Collect Particulates And The Glass Cylinder and PUF Used To Collect Vapors

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch020

magnehelic gauge hooked to a venturi. The sampler is equipped with an elapsed-time indicator to provide actual sampling time and a seven-day timer to allow the sampler start and stop time to be pre-set.

The two-stage module of the PUF sampler enables determination of suspended particulate matter concentrations by pre- and post-weighing of the four inch glass-fiber filter. Because relative differences in suspended particulate matter are of concern, this evaluation can be performed immediately upon the collection of the samples and can be used to aid site personnel in the assessment of dust suppression techniques and evaluation of meteorological conditions. This technique for the determination of suspended particulate matter concentrations should not be directly compared to national ambient air-quality standard for Total Suspended Particulate (TSP) because the filter size, sample flow rate, and sample volume do not conform to the sampling procedures outlined in the Code of Federal Regulation 40, Part 50.11, Appendix B for TSP sampling, however, it does provide accurate results.

Quality Control and Quality Assurance

The sixth step in the design of an air monitoring survey is the development of a quality assurance and quality control plan. This plan should include specific sampling procedures, calibration procedures and frequency, sample custody, analytical procedures, data reduction, internal quality control checks, performance and system audits, completeness, and corrective action.

Data Management and Reporting

The final step in the development of an air monitoring survey is determining the methodology to be used for data management and reporting. Detailed evaluation of this step is beyond the scope of this paper; however, data management and reporting should be developed to meet the overall objectives of the air monitoring survey which were clearly stated in step one.

Sampling Method Validation

To add validity to the air monitoring survey, it is important to know the effectiveness of the sampling and analytical technique used for the specific compounds in question. Questions as to the effectiveness of the PUF sampler for collection of 2,3,7,8-TCDD have come up during the course of its use. Table 1 illustrates the results of a study conducted to investigate the adsorption efficiency of the PUF sampler for dioxin. This study consisted of spiking known amounts of dioxin (2,3,7,8-TCDD) in solution (approximately 100 nanograms) on the 1st stage filter of a PUF sampler. Three individual runs were performed with sample durations of 17.5 min., 144 min., and 24 hours. The filters and the PUF plugs were analyzed separately to evaluate potential dioxin breakthrough. The average percent recovery of dioxin for the three samples was 112.5% with a standard deviation of 1.3%. The analytical recoveries of dioxin

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch020

were consistant for the three samples. Recoveries were slightly high but within the expected precision of the analytical method. The decreasing amount of dioxin found on the filter and the increasing amount of dioxin found in the PUF with increasing sample duration, in conjunction with consistant overall recovery percentages, indicates that dioxin is being air stripped off the filter and being adsorbed in the PUF with no observed breakthrough.

Table 1
Dioxin Method Validation Study Results

Run #	Duration (min)	Volume (m^3)	Relative Filter (%)	Recovery PUF (%)	Total Recovery (%)
1	17.5	4.66	100	0.0	111
2	144	46.9	96.5	3.5	113.3
3	1440	480.8	81.8	18.8	113.2

The above described study was also performed to evaluate the effectiveness of the PUF sampler for collecting 2,3,7,8-tetrachlorodibenzo-p-furan. As is shown in Table 2, the overall furan recoveries were not as consistent as the results for the dioxin but were increasing with run duration. This increase indicates that the variation is not due to breakthrough but possibly due to analytical variation. One interesting difference in the furan results is that the furan appears to air strip off the filter much more readily than dioxin. During the 17.5 minute run almost 40 percent of the furan placed on the filter had migrated to the PUF. Ninety percent had migrated from the filter to the PUF after 2.4 hours, and 100 percent after less than 24 hours.

Table 2
Furan Method Validation Results

Run #	Duration (min)	Volume (m^3)	Relative Filter (%)	Recovery PUF (%)	Total Recovery (%)
1	17.5	4.66	62.7	37.3	81.6
2	144	46.9	10.2	89.8	87.3
3	1440	480.8	0.0	100	102.2

<u>Sample Screening to Reduce Cost</u>

Sample screening techniques, which provide high quality data by enabling low cost routine analysis of all samples

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch020

and a logical procedure by which to determine samples to be analyzed for dioxin, are important and cost effective. A majority of the remedial activities now in progress and planned for in the near future involve the removal of dioxin contaminated soil and debris. Based on the nature of these sites, it seems possible that migration of the contamination, and therefore potential exposure due to remedial activities, may be related to suspended particulate concentrations. Determination of particulate matter concentrations can be done quickly and at very low cost. By analyzing for suspended particulate matter concentrations at all monitoring stations and analyzing on-site wind data, an upwind or background sample can be identified. This value can be subtracted from all the samples collected to determine the above background particulate matter concentrations. These values should represent the concentrations due to activities on the site. After 20-30 samples have been analyzed, it is expected that a relationship between above background particulate matter (PM) concentration and TCDD concentration may be established. It is expected that the concentration of TCDD in airborne particulate matter will be quite variable depending on factors such as the level of contaminant in the soil being removed, the amount of dust attributable to other sources, the particle size being suspended (function of meteorological conditions and on site activity), and whether vapor phase dioxin is evaporated from exposed contaminated soil. Even though the expected dioxin level in airborne particulate matter may be variable, this methodology may prove useful to establish an upper bound or maximum concentration of dioxin measured in airborne particulate matter. This data can be used to screen samples for dioxin analysis. Dioxin analysis would then be performed only on those samples with PM levels that exceed a predetermined threshold level, below which previous results indicate that dioxin would be nondetectable. This type of evaluation should be site specific, and may not be applicable in some cases.

Figure 3 is a graph showing the relationship between particulate matter and dioxin concentrations at one site. A total of 75 samples were collected at this site with 9 of the highest above background particulate matter concentrations being analyzed for dioxin. Of these 9 samples, four showed positive results for dioxin. The data is limited; nevertheless, there appears to be a trend of increasing dioxin concentration with increasing particulate matter.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch020

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch020

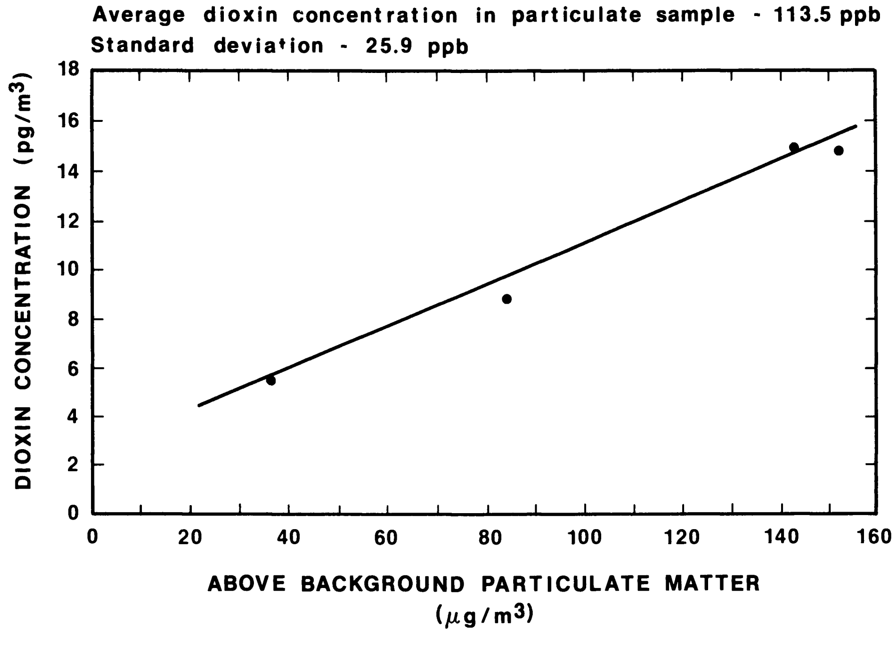


Figure 3. Trend Between Particulate Matter Concentration and Dioxin Concentration At One Ambient Air Sampling Site

Literature Cited

1. Esposito, M. P., Drake H. M., Smith, J. A., Owens, T. W., "Dioxins: Sources, Exposure, Transport and Control," Volume I, Report No. EPA-600/2-80-156, Industrial Environmental Research Laboratory, Office of Research and Development, U. S. EPA, Cincinnati, Ohio 45268 147-199.

2. Noll, K. E. and Miller, T. L. Air Monitoring Survey Design, Ann Arbor Science Publishers, 1977.

3. Lewis, R. G. and Jackson, M. D., "Modification and Evaluation of High Volume Air Sampler for Pesticides in Semivolatile Industrial Organic Chemicals," Analytical Chemistry, Vol. 54, No. 3, PP. 592-594, (March, 1982).

4. Lewis, R. G., "Monitoring Strategy for 2,3,7,8-Tetrachlorodibenzo-p-dioxin in Ambient and Source Related Air", Appendix D, Research Triangle Park, N. D., July 21, 1983.

RECEIVED November 25, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch020

Chapter 21

Remediation of a Dioxin-Contaminated Surface Impoundment

Ray K. Forrester[1], Leland Marple[2], and C. P. Carson, Jr.[3]

[1]Syntex Agribusiness, Inc., Syntex (USA), Inc., P.O. Box 1246 SSS, Springfield, MO 65805
[2]Syntex Analytical and Environmental Research, Syntex (USA), Inc., 3401 Hillview Avenue, Palo Alto, CA 94303
[3]Carson-Mitchell, Inc., 601 North Glenstone, Springfield, MO 65801

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch021

The complete remedial plan for cleanup of an unlined surface impoundment containing dioxin and solvent contaminated waste contains a brief history, preliminary sampling, pertinent scientific studies, development of a plan, site preparation, excavation of dioxin and solvent contaminated sludges and soils, on-site storage of wastes and ultimate disposal plans. Special emphasis is given to techniques for removal of dioxin from wastewater streams and a method of dewatering organic chemical sludge. The design and permitting of an on-site dioxin storage facility is described. Environmental and personal monitoring, safety, special protective equipment, decontamination procedures and other general considerations are briefly discussed.

In 1982, Syntex Agribusiness, Inc. (Syntex) ceased use of an unlined surface impoundment (Lagoon) at its Springfield, Missouri plant site. Shortly thereafter, the company began RCRA closure of the Lagoon. Early in the closure process, a series of samples were taken from the sludge deposited in the Lagoon's bottom and these samples were submitted to exhaustive chemical analysis. As soon as the results of the chemical analysis became available, Syntex sent the results to the United States Environmental Protection Agency (USEPA) and the Missouri Department of Natural Resources. The analysis of the sludge showed significant levels of common plant solvents and low parts per billion levels of 2,3,7,8 tetrachlorodibenzo-p-dioxin (dioxin). Syntex then began a removal of the standing water in the Lagoon to reduce the hydrostatic pressure (head) which might force contaminants into the ground water. The standing water in the Lagoon was processed through activated carbon, analyzed to verify that it contained no detectable dioxin, and discharged to the municipal treatment system. This removal and treatment of the standing water was done on a continual

0097-6156/87/0338-0278$06.00/0
© 1987 American Chemical Society

basis until the sludge from the Lagoon was excavated in the fall of 1985. Simultaneous to the removal of the standing water, a plan for assessing the Lagoon began to be developed.

Plan Development

A series of monitoring wells was installed on the plant site around the perimeter of the Lagoon to monitor the alluvial groundwater. Also, a series of near surface bedrock wells was installed in the bedrock (less than 75 feet in depth) to monitor the upper most aquifer.

As previously mentioned, chemical analysis of samples of the Lagoon sludge showed relatively high levels of several solvents and other organic chemicals. Based on these results and the low levels of dioxin previously determined, it seemed desirable to remove these chemical sludges to eliminate a future potential point source of contamination to the groundwater.

The Lagoon was located immediately adjacent to one of the plant's production buildings (approximately 15 feet away) and the plant's wastewater aeration basin. The foundation of the production building and the wall of the wastewater aeration basin was of particular concern since the excavation of the Lagoon sludge could induce caving of the adjacent soil embankments and put these structures at risk. To assess this potential hazard, a series of core borings was made around the Lagoon perimeter. These soil borings were also analyzed for dioxin and various solvents. Soil properties adjacent to these structures were determined to be particularly poor and the chemical analysis of these soils indicated the presence of significant quantities of toluene and xylene. No dioxin was found in any of the borings. A close study of the analytical results and plant history showed that much of the soil contamination was due to an abandoned plant sewer which was located between the Lagoon and the production building. This sewer had evidently leaked for some time and had saturated this area with solvents. This new data required plans which were a significant modification of the relatively straightforward Lagoon excavation that had been contemplated.

In order to excavate the Lagoon and protect the structures mentioned above, a procedure called "benching" was devised. Benching consists of removing a quantity of soil adjacent to the Lagoon and controlling the slope of this excavation to produce an approximately 1.5 to 1 slope. A slope of 1.5 to 1 is not as likely to suddenly fail and endanger the structures. This procedure prevented non-dioxin contaminated soil from falling into the chemical sludge and also allowed the excavation of the majority of the solvent contaminated soils along the path of the abandoned sewer.

The final work plan, which began with various site modifications, followed by benching, sludge excavation, sampling, installation of a french drain system, backfilling, and capping was then developed, reviewed by MDNR (lead agency at the site) and USEPA, and approved in August of 1985. Numerous site preparations had to be implemented prior to commencement of the actual Lagoon remediation.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch021

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch021

Site Preparations

Water Treatment Process. Prior to the development of the final plan, certain site preparations were required. A long term means of efficiently processing contaminated water had to be developed to continue to treat rainfall and infiltration to the Lagoon. During analytical work, it was discovered that all dioxin contamination of this water was due to attachment of the dioxin to suspended solids which, when filtered, removed all of the dioxin contamination in the water. Based on this discovery, a very efficient alum flocculation process was developed to remove suspended dioxin bearing particulates prior to further processing by activated carbon columns. Dioxin removal prior to carbon treatment gave extra capacity for organics removal. Furthermore, the spent carbon could be returned to the manufacturer for regeneration since it was no longer contaminated with dioxin. The carbon was then reused for further organics removal.

Storage Facility. Prior to the finalization of the remedial plan, it was determined that the excavated sludge contaminated with TCDD would need to be stored on site until an ultimate disposal method could be developed. Once measurements were made to assess the quantity of contaminated sludge (approximately 800 cubic yards), an on-site storage facility was designed and built. Plan specifications and actual installation were approved and monitored by MDNR and USEPA. The facility was sited after geotechnical borings were conducted. The facility which is approximately 36 feet wide, 120 feet long, and 8 feet deep, was constructed with one foot thick reinforced concrete walls and floor. Three equal-sized, sloped segments of the facility were drained to their own sump. The storage facility was installed over compacted clay with an underdrainage system connected to three sumps corresponding to the three segments of the facility. A second underdrainage system was then installed inside the concrete facility and the entire facility lined with 100 mil. high density polyethylene. Perforated pipes were installed at each sump to allow water to be removed from the facility once the sludge was in place. The bottom of the stand pipes was held in place with gap-graded concrete and the entire stand pipe was covered with filter fabric. The facility was then covered with pre-stressed concrete roofing panels. In the bottom of the facility a six inch layer of spent, activated carbon was installed and then covered with a geotextile membrane to allow a drainage area and to further protect the liner during filling. This storage facility was added to the plant's RCRA permit, a closure and inspection plan was written, a liner compatibility study was performed and the liner was dye tested and certified to be leak free prior to sludge being placed into the storage facility. This facility was designed to become eventually an equalization tank for the plant's wastewater treatment system once the sludge is removed for ultimate disposal and the structure is decontaminated.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch021

Staging Pad. A soil staging pad was constructed adjacent to the Lagoon to accept the non-dioxin, solvent contaminated soil for temporary storage prior to shipment to a hazardous waste landfill. This staging pad was constructed with a one foot high curb and built from reinforced concrete. The pad was situated on top of a 30-mil. high density polyethylene liner with an underdrainage system installed in between the liner and the pad. The pad was covered with a steel frame and canopy and has since been enclosed to provide a RCRA-approved drum storage area for hazardous waste. A closure and inspection plan has been developed for the storage area and the area has been added to the plant's RCRA permit.

Crane Pad. Along one side of the Lagoon a pad for the 100-ton crane was constructed by benching approximately 3 feet of soil and filling the area with shot-rock with a finish of small gravel on top to facilitate foot traffic. This pad allowed the large, tracked crane to maneuver the entire length of the Lagoon to enhance the excavation process.

Hopper and Conveyors. A hopper was built and located at one corner of the crane pad with a concrete wash down area under the hopper. The hopper was used to remove large debris on its bar grate and then used to fill the mixing trucks. It was designed with a pneumatically operated dumping device to assist the cleaning of the bar grate and a pneumatic slide gate to load the mixing trucks. An inclined conveyor was installed at one end of the storage facility and a horizontal conveyor installed on the top of the storage facility which was fed by the inclined conveyor. Both conveyors were belt-type conveyors designed to convey and place concrete. The bottom side of each conveyor was enclosed with a drip-catcher to prevent the return side of the belt from contaminating the work area. Additionally, the inclined conveyor's loading hopper was placed in a separate basin to catch any spills during the loading operation. The horizontal conveyor was designed with a diverter device which allowed the conveyor to be unloaded at any point along its length. This feature was used to place the sludge uniformly into the storage facility through holes sawed into the concrete roofing panels.

Finally, a large 100-ton crane fitted with a clam shell bucket was leased, along with a small track-type front loader. Two used, standard 10 cubic yard concrete trucks were acquired to mix and transport the sludge. A backhoe and other miscellaneous equipment which have previously been used in other remediation activities supported the cleanup. Once all the site preparation activities were completed the site cleanup could begin.

Site Cleanup

Sludge Treatment. The Lagoon chemical sludge was very waxy and did not readily give up entrained water. A pretreatment process was developed by our laboratory which allowed this sludge to be readily dewatered. The result was conversion of this difficult-to-transport material into a relatively dry, sandy textured material. The

pretreatment process involved the addition of a small quantity of calcium oxide followed by vigorous mixing. A slaking process then occurred which rendered the waxy sludge a fluid slurry which, upon standing, readily dewatered in place.

Dewatering. Geologic and hydrologic analyses conducted by our consultant suggested that a groundwater mound existed in conjunction with Lagoon ponding. Such a mound was a concern because of possible (a) Lagoon slope instability prior to dissipation of soil pore pressure and (b) excessive recharge to the Lagoon during sludge excavation. Recharge rates up to 100 gallons per minute were projected.

Initial efforts focused on removal of free water from the Lagoon. Standing water resulting from rainfall was regularly pumped to the treatment process. For a period of time prior to excavation, dewatering of sludge was attempted via pumping from an excavated sump in the northwest corner of the Lagoon. Due to the naturally high water content of the sludge and the absence of monitoring wells directly adjacent to the Lagoon, verification of mound existence/dissipation was not possible.

It was generally thought that groundwater inflow to the Lagoon would be greater through the floor than through the sideslopes because of the relative permeabilities of the materials. In order to approximate the amount of inflow to be expected, a percolation-type test was devised. For this test, a 66-inch diameter corrugated metal pipe, ten feet long, was placed vertically in the Lagoon. The clamshell bucket was used to drive the pipe to refusal in the Lagoon floor. Following evacuation of sludge and water from the pipe, the unit recharge rate through the floor would be quantified and projected for the total Lagoon area. This procedure found that water did not rise to a level within the pipe but, rather, stabilized within an inch of the Lagoon floor. These results suggested that; (a) groundwater inflow would not be a significant problem during excavation of sludge and (b) the Lagoon floor lay above the shallow water table level.

These observations held true throughout the course of sludge excavation. No groundwater problems were encountered, although surface water from abnormally high rainfall (20.31 in. actual versus 8.64 in. normal for the period 10/1/85 through 12/31/85) greatly increased the quantity of water handled by the water treatment system.

Benching. In light of previously discussed soil stability problems adjacent to the Lagoon, several precautionary measures were implemented in conjunction with closure activities to ensure slope stability. These measures included:

a) Benching of Lagoon slopes. The top Lagoon slope was excavated to a depth of two to four feet to produce a bench approximately ten feet wide. This action relieved slope surcharge (reduced the driving force) and provided a surface to contain upper slope sloughing. Solvent contaminated soil adjacent to the production building was placed on the staging pad for future disposal.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch021

b) Construction of a crane pad. A 30 ft. X 90 ft. area adjacent to the Lagoon was excavated to a depth of two feet and backfilled with 4 - 6 inch crushed limestone. The crushed limestone was topped with a 6-inch gravel layer to facilitate foot traffic. This pad provided a solid working surface for the crane and distributed crane loads to an acceptable level. Excavated soils for the crane pad were free of contamination and were placed on the clean fill stock pile to be used as back fill once the Lagoon was excavated.

c) Monitoring of Process Building. Due to the close proximity of the process building to the Lagoon, survey monuments were established along the east wall and monitored daily for displacement. No such displacement was observed.

d) Protection of completed slopes. Following excavation of sidewalls, polyethylene sheeting was placed on finished slopes to prevent subsequent contamination and provide protection from rainfall.

e) Visual inspection of slopes. During the course of Lagoon closure, slopes were inspected daily for any signs of slope instability.

Lagoon slopes performed satisfactorily during the duration of the project. Although abnormally high rainfall occurred, erosion/sloughing of slopes was minimal.

<u>Excavation Sequence</u>. Excavation commenced with scraping of exposed Lagoon slopes above the sludge level to a depth of six inches. This material was considered potentially contaminated and added to the Lagoon. After the slopes were scraped, benching of adjacent non-dioxin, solvent-contaminated soil was performed by the front loader. This material was transported by clamshell and placed on the staging pad.

After these activities, excavation of sludge began. The sludge was excavated with the clamshell and placed in the elevated hopper. Materials not passing the bar grate on top of the hopper and other materials removed directly from the Lagoon were drummed and stored on the staging pad. Prior to discharging the hopper, a measured quantity of calcium oxide was added to hopper. The contents of the hopper was then dumped into the concrete mix truck and vigorously mixed for approximately five minutes. Upon mixing, the truck was moved to the inclined conveyor and contents discharged to the conveyor loading hopper. The inclined conveyor then fed the horizontal conveyor which was diverted to a series of holes cut into the storage facility roof.

This procedure was utilized successfully until the majority of sludge was removed from the Lagoon. At this point, however, the shallow depth of sludge, approximately six inches, and greatly decreased percentage of solids, made clamshell excavation

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch021

inefficient. To complete removal of sludge, a quantity of calcium oxide was added to the Lagoon and mixed with the track loader. The track loader was then used to pile the thickened material for clamshell excavation. This operation was repeated until all sludge was removed from the Lagoon. A section of the removable concrete roof was set aside to allow the clamshell to drop the treated sludge directly into the storage facility.

After the sludge was removed, the Lagoon bottom and slopes were scraped until no visible contamination remained. A rubber-tired backhoe was committed to the Lagoon to supplement the track loader for this activity. The Lagoon bottom was found to be firm and readily supported vehiclular traffic. No groundwater inflow to the Lagoon occurred. Excavated material was again piled by the two loaders and clamshelled directly to the storage facility.

Once the contaminated materials were placed inside the storage facility, the treated sludge rapidly dewatered to the three sumps. Water was pumped from the sumps in the storage facility as the dewatering occurred. This process will continue until no water remains in the sumps. Any water pumped from the facility is treated, analyzed and discharged to the municipal sewer system.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch021

Sampling

Following sludge excavation, the Lagoon bottom was sampled in accordance with the approved sampling protocol. Eighteen discrete samples were obtained and composited into three samples representing the north, middle, and south sections of the Lagoon floor. Discrete sample coordinates and elevations were tabulated. The highest levels of dioxin and total organics were 0.14 ppb and 875 ppm, respectively. The detected levels of dioxin are well below the 1 ppb level recognized by CDC as acceptable for residential exposure with one hundred percent surface contamination. The remaining organics will be addressed in the groundwater remediation program.

French Drain

To recover any remaining organic contamination from the adjacent soils a french drain system was installed in the bottom of the excavated Lagoon.The french drain design used a granular layer over the entire Lagoon floor, allowing capture of water migrating upward through the floor (such as with naturally fluctuating watertable conditions) in addition to any rare seepage entering through the Lagoon walls. In conjunction with the drainage layer, a stainless steel well was constructed at the low point on the Lagoon floor to accommodate monitoring and recovery of fluids. Since the well could draw fluids from all directions, no supplementary piping was required.

The large crushed rock from the crane pad was pushed into the Lagoon to provide the porous drainage area of the french drain. In order to ensure absolutely free drainage to the monitoring/recovery well, a 24-inch wide perimeter trench was excavated in the crane pad

material and backfilled with clean 5/8-inch crushed limestone. The monitoring/recovery well, sited in the southeastern portion of the Lagoon, utilized an additional quantity of 5/8-inch crushed limestone as gravel pack. Following construction of the french drain, geotextile fabric and a one-foot thick layer of base course were placed above the porous drainage area for protection during backfill.

Backfill

Backfill of the Lagoon proceeded with soil earlier stockpiled on site. This material, derived from offsite construction excavations, consisted of dark red residual clay native to the Springfield area. Laboratory analyses certified this material to be free of dioxin and other contaminants.

To ensure that the required cap permeability (1.0×10^{-7} cm/sec) could be achieved, laboratory testing of the remolded virgin soil was conducted. These results confirmed that the required permeability could be achieved utilizing virgin soil compacted to approximately 95% Standard Proctor Density.

The backfill was placed in a maximum of one-foot lifts and compacted to approximately 95% Standard Proctor Density using the track loader and rubber tired backhoe. This procedure effectively produced a 10-foot thick seal exhibiting 1.0×10^{-7} cm/sec permeability and precludes the possibility of appreciable settlement. Field compaction tests were performed during the backfilling to assure that proper compaction was achieved.

Once the backfill was completed it was graded to a two percent slope for rapid stormwater drainage. The final graded area was then covered with gravel.

CONCLUSION

This paper described the process through which a team of environmental engineering, geotechnical engineers, analytical chemists, and other scientists addressed the cleanup of a dioxin and solvent contaminated site. The remediation program used here was successfully completed without posing a hazard to the community or the workers, yet it was completed in a timely manner at a small fraction of the cost normally reported for cleaning less complex sites. The entire excavation was completed in 21 days using a crew of six remediation workers. Every structure and piece of equipment is designed to provide a future long term use or can be decontaminated through proven methods and resold to minimize cost. All contaminated materials were handled and stored in bulk to eliminate costly bagging or drumming of the materials. This bulk handling of materials can dramatically reduce cleanup costs.

Experience, planning, training and careful project implementation provide the ingredients for a remediation project that meets or exceeds all environmental and budget goals. This remediation package should be a useful model for regulatory agencies and firms faced with the evaluation and cleanup of similar lagoons or landfills contaminated with dioxin and/or organic chemicals.

RECEIVED November 25, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch021

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch022

Chapter 22

Removal of 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin from Waste Water and Well Water

Coagulation and Flocculation with Aluminum Salts

Leland Marple, Dale Dei Rossi, and Lewis Throop

Syntex Analytical and Environmental Research, Syntex (USA), Inc., 3401 Hillview Avenue, Palo Alto, CA 94303

At various times, water in a wastewater lagoon contained several parts per trillion 2,3,7,8-tetrachlorodibenzo-p-dioxin (dioxin). A process was developed for the removal of trace amounts based on coagulation and flocculation with aluminum salts. On site treatment utilized a batch process in which suspended solids were coagulated with 1200 mg/l aluminum sulfate. A polymer modified alum flocculation process was developed for the removal of dioxin from well water. The modification of the floc surface by the adsorption of an uncharged, high molecular weight polymer intensifies the adsorption of dioxin on the floc.

One of the major problems involved in the remediation of the Dioxin contaminated surface impoundment (lagoon) discussed earlier (1) was the treatment of the water to insure complete removal of any 2,3,7,8-tetrachlorodibenzo-p-dioxin (Dioxin) that may be present. Initially, suspended matter was removed by filtration through ground corncobs, and residual organics were removed by treatment with activated carbon. Although this treatment was effective, the processing time per batch coupled with groundwater seepage and periods of heavy rainfall prolonged the drainage of the lagoon for over a year.

The selective removal of very low levels of dioxin from large volumes of process wastewater has become a concern to a number of companies and municipalities. Very little has been reported on how dioxin behaves in conventional water treatment processes (2-3). The work of Thebault, Cases, and Fiessinger suggested that alum flocculation would be marginally effective for the removal of dioxin. Our need to remove up to several parts per trillion dioxin from water in the lagoon prompted us to evaluate flocculation as a faster and more cost effective treatment for dioxin removal.

0097-6156/87/0338-0286$06.00/0
© 1987 American Chemical Society

As it turned out, dioxin was readily removed by alum flocculating in the presence of suspended organic matter. The successful treatment of lagoon water prompted work on two related problems. Firstly, we needed to find a way to dewater the asphaltum like lagoon sludge, and secondly, we needed to find a way to treat wellwater, having little suspended solids, to remove dioxin that might be present. This paper describes our experimental work on these problems and application of our findings to treatment at the plant site.

EXPERIMENTAL

The amount of aluminum needed to produce rapid flocculation of lagoon water with a suspended solids content of about 3200 ppm was determined by 100 ml jar tests. A 0.002 uCi spike of 14-C dioxin (Kor Inc.) was added to the water and equilibrated for 2 hours before flocculation. Ammonium sulfate, 500 ppm, was added to increase the ionic strength and promote coagulation. The buffer capacity of the water was sufficient to precipitate the aluminum added, and the final pH of the water was about 8. The amount of dioxin spike remaining in solution was determined by centrifuging the sample, withdrawing a 50 ml aliquot of the clear supernatant and extracting the dioxin with hexane. The hexane extract was reduced in volume in a rotary evaporator, then transferred to a glass scintillation vial along with 5 ul of n-tetradecane. The extract was reduced to near dryness with a stream of nitrogen, diluted with scintillation fluid, then counted in a Packard TriCarb scintillation counter.

The flocculation process was scaled up to treat 20,000 gallons per batch. The supernatant from each batch was analyzed for dioxin, then treated with activated carbon for removal of residual organics The settled floc was returned to the lagoon for dewatering.

The coagulation of lagoon sludge by lime was followed by a simple jar test. A 40 g sample of sludge was mixed with water to bring the volume to 200 ml. Increments of 0.10 g calcium oxide were added and mixed well. The mixture was allowed to settle for 5 minutes after each addition, and the extent of coagulation estimated from the clarity of the supernatant. The dewatering of coagulated sludge was demonstrated by building a 4 inch by 6 foot column in 12-6 in. increments over a period of 9 days. The lime was added directly to the sludge and mixed by inversion of the container. The coagulated sludge was retained by a 2 inch bed of activated carbon held in place by a perforated stainless steel screen. Water draining from the column was pooled until the last addition was made, then it was collected on a daily basis.

The removal of dioxin from water by modified alum flocculation was monitored by the disappearance of a 0.08 uCi spike of 3-H labeled dioxin (obtained from A. Poland, repurified by silica column chromatography) in jar type experiments. A conventional uncharged Garratt-Callahan polymer #7882 was used for floc modification. The labeled dioxin spike, dissolved in hexane-methylene chloride, 80:20, was introduced into 200 ml of water in several ways. The spike was added directly to the water, it was added to water containing the

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch022

modifying polymer, and it was evaporated as a film on the inside of the beaker used to hold the water sample prior to flocculation. After the alum was added, the water was flash mixed for two minutes, then stirred slowly for 5 minutes while the floc formed. The floc was allowed to settle for 15 minutes, then the supernatant was analyzed for dioxin by withdrawing a 50 ml aliquot, extracting the dioxin with hexane, and working up the extract as described above.

RESULTS/DISCUSSION

The removal of 14-C dioxin from lagoon water by flocculation with varying amounts of aluminum followed by centrifugation is illustrated in Table I.

Table I. Removal of 14-C Dioxin spike from Lagoon Water by Flocculation with Aluminum Salts

$AlCl_3$	44 ppm Al	(u)%
	87	99.8
	87	99.7
	100	99.7
	130	99.6
Alum	89 ppm Al	98.5
	89	99.5

(u) unmeasured as coagulation was incomplete

The removal was calculated from the counts remaining in solution compared to the counts added to the system. Although there is very little information in the literature on removal of chlorinated organics by alum or lime flocculation, our results are consistent with available data. For example, Saleh, Lee and Wolf (4) removed about 95% of combined DDT isomers from an activated sludge effluent upon coagulation/flocculation with a 23 ppm/165ppm lime/alum mixture. One would expect a greater adsorption of dioxin compared to DDT, owing to the lower water solubility of dioxin.

Scale-up of the treatment process proceeded smoothly. Table II summarizes the levels of dioxin found in the supernatant for batches processed at the plant site.

While low levels of other soluble organics, such as mixed xylenes, ethylbenzene and toluene, are also adsorbed by the floc, data are not available for their removal at the site.

Since the major difference, in principle, between water in the lagoon and the sludge in the bottom was the peptized solids content, coagulation appeared to be the logical route for sludge dewatering. This was demonstrated by suspending a sample in water, and titrating suspension with alum until the solids coagulated. Since coagulation with lime is generally more cost effective than alum, we determined the amount needed by a simple jar test. Although the coagulation appeared to be complete at 1% lime, we allowed for variation in sludge composition by using a 25% excess

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch022

Table II. Removal of Dioxin from Lagoon Water at Plant Site by Alum Flocculation

Batch #	Influent	Effluent
021-R-1	3.1 ppt	0.35 ppt
021-R-2	*	0.17
022	*	0.20
022		0.21
023		0.17
024		0.16
025		0.27
026		0.93
027		0.70
028-1		0.42

*undetermined, but from the same source as 021-R-1

for the preparation of a dewatering test column. The total weight of the treated sludge put in the column was 22.89 Kg. Water drained from the column over the course of sludge addition amounted to 7.00 Kg, representing 98% of the water ultimately drained from the bed. Dioxin could not be detected in the pooled water sample, nor could it be detected (< 0.6 ppt) in water subsequently drained from the sludge bunker at the plant site.

We did not expect that the removal of dioxin from well and runoff water by alum coagulation and flocculation would be effective, as there is little or no suspended organic phase to act as a carrier for dioxin. This problem was solved by adding a high molecular weight polymer that would be adsorbed to and carried down with the floc. Partition of dioxin into the polymer is so favored that parts per million levels of polymer should remove most of the dioxin. The results of jar tests to find the optimum level of polymer, at a fixed 100 ppm level of aluminum are given in Table III.

Table III. Removal of 3-H Dioxin Spike from Tap Water by Polymer Modified Alum Flocculation

Conc. Polymer, #7882	% Dioxin Removed at 100 ppm Al
12.3	86.1
18.5	92.8
18.5	89.0
24.6	68.0
24.6	73.8
30.8	84.6

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch022

If one assumes that the partition of dioxin between polymer and water is equivalent to the partition between octanol and water, the amount of dioxin in solution should drop by at least 94%. The penalty for this process is an additional solids waste loading of about 2.5 pound per 1000 gal. treated water.

The effect of introducing dioxin into the system by different routes is given in Table IV.

Table IV. Summary of Dioxin Removals by Modified Alum Flocculations

Method	% Initial Spike Dissolved	% Soluble Spike Removed	Polymer/Al Ratio ppm
Solid	65	85	18/100
Dioxin Residue		25	0/100
Dioxin to Water	30	86	12/100
with Polymer		93	18/100
Dioxin to Water	60	89	18/100
		90	18/80
		75	18/80

The various routes were used to avoid artifacts arising from adsorption at surfaces in the equilibrating system. It is clear from these data that the removal of dioxin by a 12-18 ppm polymer modified alum floc was very close to 90%. Verification of the removal of dioxin by the floc was obtained by recovering the floc and measuring the tritium activity. This was needed in order to rule out the possibility that the polymer merely enhanced the loss to the system. As it turned out, the amount of dioxin that was trapped by the floc was greater than that calculated from the decrease in soluble dioxin in solution, so some of the dioxin originally lost to the system was recovered by the floc. Consequently, scale up of this process at the plant site has the potential of being more efficient at removing dioxin from water than we predict from laboratory experiments.

LITERATURE CITED

(1) Forrester, R.F., Marple, L.W. Carson Jr., C.P., in "Solving Hazardous Waste Problems: Dioxins," Exner, J.H., Ed.; ACS Symposium Series No.___, American Chemical Society, Washington, D.C. 1987, P.____.

(2) Robeck, C.G., Dostal, K.A., Cohen, J.M., Dreissel, J.F., J. Am. Water Wks. Ass. 1965, 57, 181-199.

(3) Thebault, P., Cases, J.M., Fiessinger, F., Water Research 1982, 15, 183-189.

(4) Saleh, F.Y., Lee, G.F., Wolf, H.W., Water Research 1982, 16, 479-488.

RECEIVED November 25, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch022

Chapter 23

Comparison of Laboratory and Field Test Data in the Chemical Decontamination of Dioxin-Contaminated Soils

R. Peterson[1], E. Milicic[1], C. Novosad[1], and C. Rogers[2]

[1]Galson Research Corporation, 6601 Kirkville Road, East Syracuse, NY 13057
[2]U.S. Environmental Protection Agency, 26 West St. Clair, Cincinnati, OH 45220

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch023

A series of patented (1) processes have been developed for chemical decontamination of soils contaminated with halogenated aromatics, including polychlorinated dibenzo-p-dioxins (PCDD), chlorinated benzenes, polychlorinated biphenyls and similar materials. These processes allow reduction of PCDD levels to less than 1 part per billion (ppb) in as little as two hours at moderate temperatures and pressures.

Chemical decontamination is an alternative to thermal processing or landfilling of soils contaminated with polychlorinated dibenzo-p- dioxins (PCDD) or other aromatic halides such as chlorobenzenes or polychorinated biphenyls (PCB). Chemical decontamination, like incineration, involves changes to the chemical structure of the dioxin molecule. While chlorinated dioxins are thermally stable, they readily dechlorinate to water soluble compounds under relatively mild conditions of temperature and pressure. For example, chlorinated dioxins in oil are reduced to the ppt level within 60 minutes at 150 degrees C. by reacting them to a compound which is no longer oil soluble. In soils processing, the dioxin is dechlorinated to a water soluble form which is then leached from the soil using countercurrent extraction with water. Dechlorination also affects the toxicity of the dioxin, with dioxins containing fewer than three chlorine atoms generally showing low toxicity (2).

Process Chemistry

The proposed mechanism for these reactions is shown in Figure 1 using 2,3,7,8 tetrachlorodibenzo-p-dioxin as an example. An alkali metal hydroxide, usually potassium hydroxide (KOH) is treated with an alcohol or glycol such as polyethylene glycol 400 (PEG 400) to form an alkoxide. The alkoxide reacts with one of the chlorine atoms on the chlorinated dioxin to produce an ether and the alkali metal salt.

0097-6156/87/0338-0291$06.00/0
© 1987 American Chemical Society

The reaction may proceed to complete dechlorination, although replacement of a single chlorine is sufficient to make the reaction products water soluble. The ether formed by the dechlorination may degrade to a phenol form or may remain as the ether, depending on the reaction conditions. The processing is carried out using dimethyl sulfoxide (DMSO) as a solvent, which increases the base strength of the alkoxide. In addition, the DMSO aids in the extraction of the PCDD from the soil.

Toxicity Considerations

Chemical decontamination of soil is a two-stage process;

1. Dechlorinate PCDD to lower toxicity/ water soluble form
2. Wash excess reagents and PCDD products from soil

A major concern in this type of processing involves the toxicity of any reagents and/or reaction products which may inadvertently be left in the decontaminated soil after treatment. Some toxicity data on reagents used in the process are shown in Table I, along with comparison values for sodium chloride and 2,3,7,8-TCDD.

Table I - Toxicity of Reagents and Comparison Materials

Material	LD50, Oral-rat (3)
polyethylene glycol 400	27,500 mg/kg
dimethyl sulfoxide	17,500 mg/kg
sodium chloride (comparison value)	3,000 mg/kg
2,3,7,8-TCDD (comparison value)	0.022 mg/kg

The reagents used in this process are some five times less toxic than table salt, and roughly six orders of magnitude less toxic than 2,3,7,8 TCDD, the dioxin isomer of major concern. Polyethlyene glycol 400 is an FDA approved material for use in foods and cosmetics. Dimethyl sulfoxide is a naturally occurring material in foods such as potatoes, milk and coffee at the part per million level. Expected residual levels of these materials in soil are not expected to be a serious concern.

Toxicity testing of the converted aromatic halides is currently underway with EPA sponsorship. Structural assessment of the theoretical toxicity of the reaction products is favorable, ie the known reaction products would not be expected to show significant toxicity. Results of the Ames test for mutagenicity are negative, ie the reaction products do not demonstrate carcinogenic potential. Bioaccumulation tests also produced negative results, which is not surprising given the water solubility of these reaction products. Acute toxicity tests are currently in progress, with intial results showing low toxicity .

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch023

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch023

Process Description

The decontamination of soil proceeds in a series of six process steps.

1. Combine equal masses of soil and reagent to form a slurry
2. Mix and heat soil/reagent slurry to 100-180 °C
3. Allow to react 1-5 hours
4. Decant excess reagent
5. Wash soil 2-3 times with water
6. Discharge decontaminated soil

This process is shown in diagram form in Figure 2. All of the process steps can be conducted in a single reactor equipped with an agitator. The number of wash steps required depends on the effectiveness of each wash step and on the degree of reagent recovery required.

Results of Tests to Date

Three series of tests using dioxin contaminated soil have been conducted to date: laboratory tests at high and low rates of agitation and field tests at low agitation only. Each set is discussed separately.

Laboratory Testing - High agitation. Initial laboratory tests used 250 g. soil samples spiked to a nominal concentration of 2000 parts per billion (ppb) of 1,2,3,4-TCDD. The 1,2,3,4 isomer was used in place of the 2,3,7,8-TCDD isomer to simplify experimental and safety procedures. Tests with both isomers in liquid solution show similar reaction rates. These tests used an electrically heated 1000 mL three neck flask equipped with a reflux condenser and high torque agitator to provide a high degree of mixing.

In the initial series of experiments a fluorocarbon paddle was used with the agitator. Analysis of the treated soil samples revealed the presence of an unknown halogenated contaminant which was later determined to be partially decomposed fluorocarbon. The combination of reagent and erosion from the soil had broken down the fluorocarbon used in the agitator. This interference required some additional sample cleanup. Changing to a glass paddle solved the problem for laboratory testing.

Treated samples were analyzed by three different labs using either gas chromatography/mass spectroscopy (GC/MS) or GC/MS/MS methods. Analysis by gas chromatography alone was unsuccessful, partly due to the fluorocarbon interferences previously noted. The results of this initial testing are summarized in Table II.

These tests indicated that while reaction rates for soils were lower than those obtained in oil tests, the overall reaction times were reasonable for large scale application.

Laboratory Testing - Low Agitation. After design of the field test equipment, it became apparent that the degree of agitation obtained in the initial laboratory tests was not going to be achieved in the field. Therefore, tests were conducted at a low rate of agitation to provide a prediction of the

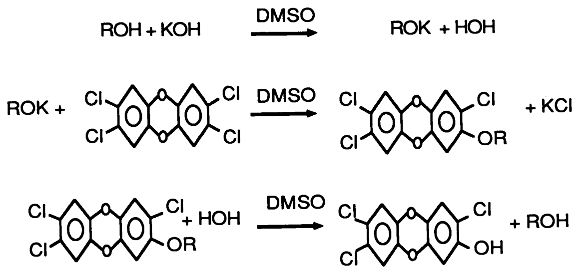


Figure 1. Proposed reaction mechanism.

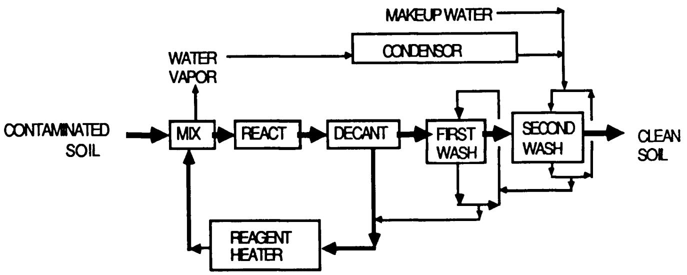


Figure 2. Process diagram.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch023

In Solving Hazardous Waste Problems; Exner, J.;
ACS Symposium Series; American Chemical Society: Washington, DC, 1987.

Table II - Results of Laboratory Testing with High Agitation
Initial Concentration 2200 ppb 1,2,3,4-TCDD

Reaction Temperature, ^oC	Reaction Time, Hours	Final TCDD Concentration, ppb
260	4	<1
150	2	<1
100	2	<0.2
70	0.5	15
70	2	<1
50	2	29
25	2	36

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch023

probable results of field testing. This testing used a flask equipped with a condenser and inserted into a heated oil bath. Agitation was provided by manually swirling the flask and contents at periodic intervals. Soil for this test was the same soil to be used for field testing, and contained "native" 2,3,7,8 TCDD. Analyses were made using GC/MS/MS techniques. The results of testing are shown in Table III.

Table III - Results of Laboratory Testing at Low Agitation, 125 ^oC
Initial concentration approximately 300 ppb 2,3,7,8 TCDD

Reaction time, hours	TCDD level, ppb
0	175
1.5	15.1
4.25	2.06
7.0	0.3

These data indicate that the reaction time for samples with low rates of mixing are on the order of 2-3 times longer than those for samples with high rates of mixing, but still well below 8 hours.

Field Test Results. Field testing for this test series consisted of a series of runs using 30 kg. soil samples taken from a dioxin site in Mississippi. Herbicide Orange had been stored at this site, with some spillage, causing soil contamination.

Test equipment for this series of tests is shown in Figure 3. A 55 gallon drum was modified by the addition of a steel plate halfway down the drum. The plate was pierced by a valve and by a tap to allow pumping of liquids out of the reservoir. The steel plate was sealed with a silicone sealant around the perimeter of the plate. Contaminated soil and reagents were added to the drum by weight. The drum was then covered and a vent line attached between the drum and a carbon filter. A heat tape was wrapped around the drum, and the entire drum was insulated with fiberglass.

The insulated drum was placed on a drum rocker and rocked from side to side to mix the soil and reagent during heating and reaction. The results of the testing are shown in Table IV.

Table IV - Results of Field Testing, 2,3,7,8-TCDD

Reaction time, hours	TCDD level, ppb	
	Initial	Final
1.0	154	37.3
2.5	356	10.7
6.5	equipment failure	

The equipment failure occurred at the seal around the perimeter of the steel plate holding the soil/reagent slurry out of the reservoir. In the 6.5 hour run this seal failed, which caused the reagent to separate from the soil and stop the reaction. As noted in the laboratory testing, the reagent is very corrosive to polymers, including fluorocarbons. Seal material selection will be studied in depth prior to scaleup of this process.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch023

Discussion of Laboratory and Field Data

The data from the three series of tests is summarized in Figure 4. Three points can be noted from these data;

1. For each reaction, an initial rate of reaction is followed by a second lower rate of reaction, vs. the single line reaction plot expected for a single order reaction.
2. The laboratory data for the high mixing case indicate a higher rate of reaction than for the lower rate of mixing. This difference is primarily in the initial reaction rate, while the secondary reaction rate is closer to that for the low mixing case.
3. The rates of reaction for field tests and for laboratory tests at low agitation rates are very similar.

The bimodal reaction rate is characteristic for soils treated by this process but not for oil treatment, in which a single line reaction plot is observed. This dual reaction rate may be due to the heterogeneity of soils. Organics on soils may be adsorbed on the surface of the soil particles, in the micropores of the soil or even wrapped up in the helical humic structures present in some soils. A bimodal reaction rate would be consistent with a process in which extraction of the dioxin from the soil into the reagent is the rate-limiting step. Extraction of dioxin from the micropores would be expected to be much slower than from the surface of the soil particles. This hypothesis is consistent with overall rate data showing that the rate of reaction is much higher for liquids than for soils, indicating an extraction limited process.

The micropore/soil surface phenomenon may also explain why the high mixing case shows a much greater difference in initial and final reaction rates than those for the low mixing case. Despite the fact that the soil for all tests came from the same site, the soils for the high mixing case were spiked

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch023

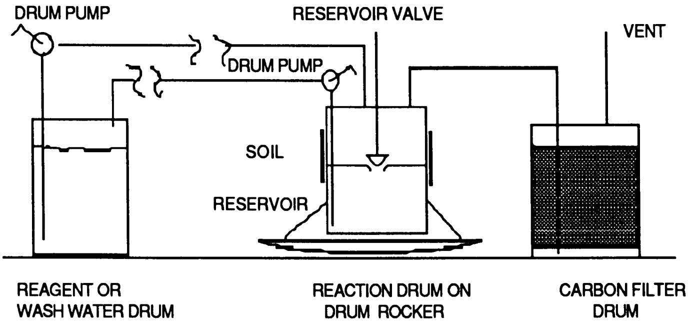


Figure 3. Apparatus for field soils processing.

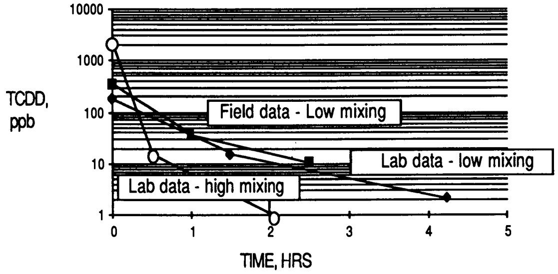


Figure 4. Results of soils processing.

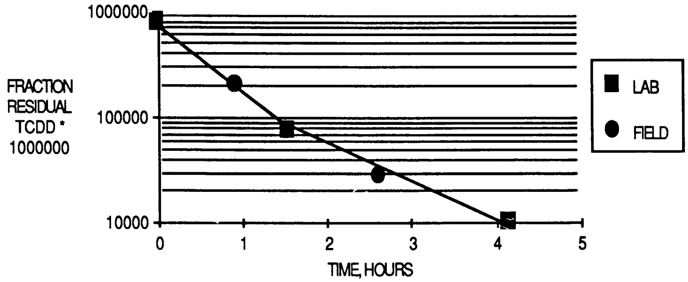


Figure 5. Comparison of laboratory and field data at low agitation.

with dioxin on the same day as the soil was processed. By contrast, the low mixing case soils had weathered for more than five years. It may be that weathering may change the micropore/soil surface distribution of the adsorbed dioxin, possibly by differential volatilization of the dioxin from the surface or by successive displacement of the dioxin by other materials.

The low mixing data for laboratory and field data show a very high degree of correlation. If placed on a normalized graph, these data fall on a single line, as shown in Figure 5. This indicates that the 100:1 scaleup of the process was successful in demonstrating that the procedure is not strongly dependent on sample size.

Conclusions

The results of this study can be summarized as follow;

1. Chemical decontamination of dioxin contaminated soils can reduce dioxin levels to < 1 ppb under laboratory conditions using either high or low rates of agitation.
2. Increasing rates of agitation yield increasing rates of reaction, although other factors may also be involved.
3. Field test data at low rates of agitation are very comparable to laboratory data at low rates of agitation.

Acknowledgments

This work has been sponsored by the United States Air Force and by the United States Environmental Protection Agency under EPA contract 68-03-321.

Literature Cited

1. Peterson, R. L. U. S. Patent 4 574 013, 1986.
2. Esposito, M. P.; Tiernan, T. O.; Dryden, F. E., "Dioxins", EPA-600/2-80-197, p. 187
3. "Niosh Registry of Toxic Effects of Chemical Substances", U.S. Department of Health and Human Services, 1981-2

RECEIVED December 12, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch023

Chapter 24

Destruction of Dioxin Contamination by Pyrolysis Techniques

Jimmy Boyd[1], H. D. Williams[2], R. W. Thomas[2], and T. L. Stoddart[3]

[1]J. M. Huber Corporation, P.O. Box 283, Borger, TX 79008
[2]EG&G Idaho, P.O. Box 1625, Idaho Falls, ID 83401
[3]Headquarters, Air Force Engineering Services Center, Research & Development Directorate, Environics Division, Environmental Engineering Branch, Tyndall Air Force Base, FL 32403

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch024

The J. M. Huber Corporation Advanced Electric Reactor (AER) pyrolysis process was field-demonstrated in a proof-of-principle test to show that dioxins in contaminated soil could be destroyed to less than 1 ppb. Testing was conducted at a former Herbicide Orange storage site at the Naval Battalion Construction Center (NCBC) in Gulfport, MS, during June 1985. Sample analysis of treated soil shows total isomer classes of tetra-, penta-, and hexapolychlorinated dibenzo-p-dioxins and tetra-, penta-, and hexapolychlorinated dibenzofurans to be less than 0.12 ppb. Herbicide compounds 2,4-D and 2,4,5-T and 2,4-di and 2,4,5/2,4,6-trichlorophenol compounds in the soil feedstock were also destroyed in the treated soil to levels below detectability. NCBC treated soil meets the Environmental Protection Agency delisting characteristic requirements. Lead and zinc are enriched in the baghouse material, which suggests that the process can be used for recovery of inorganic materials from contaminated soils that volatilize at the AER operating temperature. Test results clearly demonstrate the extremely high destruction capabilities of the AER process.

Dioxin contamination of soils has occurred throughout the world. Herbicide Orange (HO), primarily composed of two compounds, 2,4-D (2,4-dichlorophenoxyacetic acid) and 2,4,5-T (2,4,5-trichlorophenoxyacetic acid) and various esters of these two compounds, was sprayed

0097-6156/87/0338-0299$06.00/0
© 1987 American Chemical Society

as a defoliant in Vietnam during the 1960s and at Eglin Air Force Base, FL, between 1962 and 1970 (1,2). 2,4,5-T was subsequently identified as teratogenic in mice and rats, with tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) shown to be the toxic containment (3,4). During the early 1970s, unpaved roads and riding arenas in Times Beach, MO, were sprayed with salvage oil believed to have been mixed with waste material containing 2,3,7,8-TCDD in high concentrations (5). In 1976, an explosion in a trichlorophenol production plant at Seveso, Italy, caused significant 2,3,7,8-TCDD contamination to the surrounding area (6). Dioxins have also been identified as combustion products released from municipal and industrial incinerators through the combustion of chlorinated precursors present in the fuel or by reaction of organic compounds with inorganic chlorine both present in the fuel (7-12). Through long-term release, these incinerator emissions of dioxins can contaminate downwind soils to reach levels of concern to the public health. Exposure from dioxin-contaminated soil conditions as shown in these examples may place the public health at risk.

The U.S. Environmental Protection Agency (EPA) presently has no limits on dioxin contamination levels in soil. After studying the risks, however, the Centers for Disease Control (CDC) of the U.S. Department of Health and Human Services in Atlanta, GA, has concluded that residual soil contamination levels at or above 1 ppb of 2,3,7,8-TCDD in residential areas cannot be considered safe and represent a level of concern (5). Uniform contamination levels are assumed. CDC also recommends low levels for pasture lands because of food chain accumulation. However, CDC concludes that, in certain commercial areas, higher levels in soils may represent an acceptable risk to nonoccupationally exposed individuals, but that level has not been defined.

In September 1983, the J. M. Huber Corporation of Borger, TX, embarked on a program to demonstrate the capability of the Advanced Electric Reactor (AER) to detoxify 2,3,7,8-TCDD contaminated soil. This program also includes the treatment of soils contaminated with polychlorinated biphenyls (PCBs), carbon tetrachloride, and octachlorodibenzo-p-dioxin (OCDD). A field demonstration of 2,3,7,8-TCDD contaminated soil at a former HO storage site at the Naval Construction Battalion Center (NCBC) in Gulfport, MS, represents a continuation of this demonstration process. This testing was performed to demonstrate innovative technologies as part of the Air Force Environmental Restoration Program for former HO sites. A specific goal of this technology testing was to reduce the total concentration of all isomers of tetra-, penta-, and hexa-CDD and respective isomers of chlorodibenzofurans to less than 1 ppb.

Method

AER Process. The AER employs a new technology to rapidly heat material to approximately 2200°C, using intense thermal radiation in the near infrared region (Figure 1). The reactions, which can be gaseous, liquid, or solid, are isolated from the reactor core walls by means of a gaseous blanket formed by flowing nitrogen radially inward through the porous core walls. Carbon electrodes are heated and, in turn, heat the reactor core to incandescence so that the heat transfer is accomplished by thermal radiative coupling from the core to

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch024

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch024

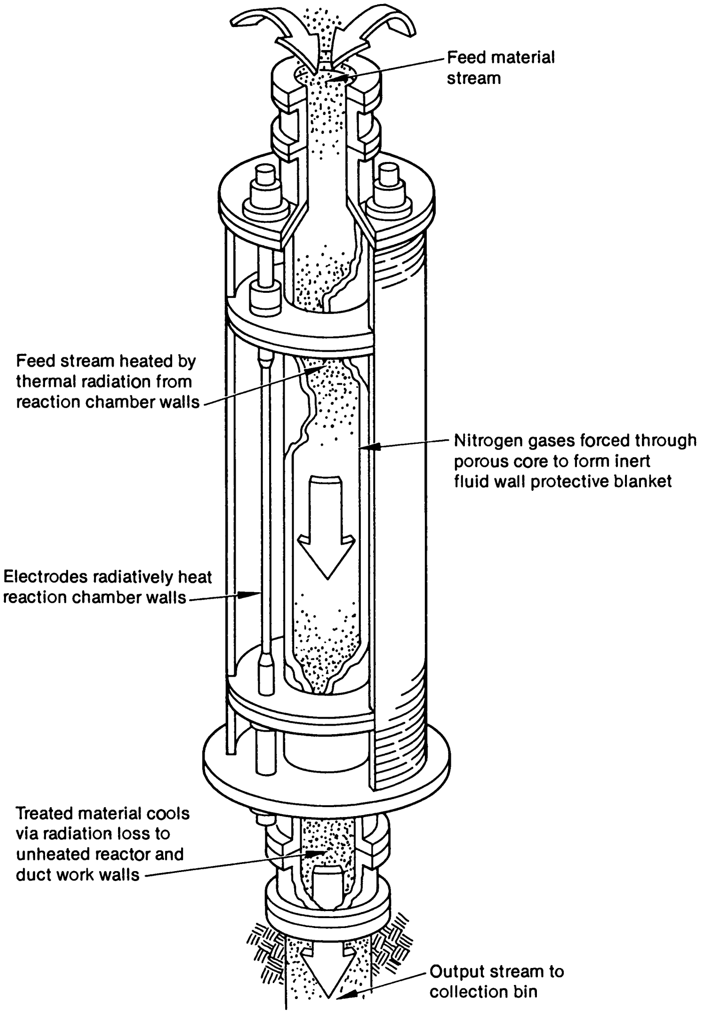


Figure 1. Advanced Electric Reactor.

the feed materials. The only feed streams to the reactor are the solid, liquid, or gaseous wastes and the nitrogen blanket gas.

Destruction is accomplished by pyrolysis rather than oxidation; therefore, typical products and by-products produced by incineration such as carbon monoxide, carbon dioxide, and oxides of nitrogen are not formed in significant concentrations. The principal products from soil-affixed TCDD using the Huber process are hydrogen, chlorine, hydrochloric acid, elemental carbon, and granular free-flowing soil material.

Figure 2 is a simplified process diagram of the AER process for solid hazardous waste destruction. The solid feed steam is introduced at the top of the reactor by means of a metered screw feeder connecting the airtight feed hopper to the reactor. Nitrogen is introduced primarily at two points in the reactor annulus. The solid feed passes through the reactor where pyrolysis occurs at temperatures between 1900 and 2500°C. After leaving the reactor, the product gas and waste solids pass through a postreactor treatment zone (PRTZ). The PRTZ provides for additional residence time but primarily cools the gas to less than 550°C before downstream particulate cleanup.

Solids exiting the PRTZ are collected in a solids collection bin that is sealed to the atmosphere. Any entrained solids in the product gas are removed as the gases enter a baghouse. Any residual organics and chlorine are removed by passing the product gas through activated carbon beds (or a caustic scrubber, if necessary) just upstream of the process stack. The clean product gas, composed almost entirely of nitrogen (some moisture), is then emitted to the atmosphere through the process stack.

An integral part of the AER design is to ensure safety of personnel and the environment when processing hazardous wastes. For example, if electrical power to the process is interrupted, the motor-driven screw feeder stops feeding solids, and within one to two seconds, all solids are purged from the reactor by gravity flow. The large amount of thermal inertia, in proportion to the feed rate, ensures continued waste destruction and safe clearing of feed from the reactor. To protect the electrodes and maintain a noncombustible environment, nitrogen purge to the reactor remains until the reactor temperature is approximately 100°C. The AER is also equipped with an automatic trip to cut electrical power in the event of electrode failure.

Huber maintains two fully equipped AERs (AER3 and AER12). The AER3, which was used for the NCBC test and is the smaller reactor, has an inside core diameter of 7.6 cm, with a heated length of 91.4 cm. The unit, installed in a trailer for mobility, is used for proof-of-concept experiments and onsite demonstrations. The AER12, which is of pilot/commercial scale (a 30.5-cm core diameter with a heated length of 365.8 cm), is used for research such as scale-up design, process engineering, economic studies, and technology demonstrations.

Site Conditions. The indigenous soil at NCBC is sand to sandy loam, intermixed with some clay. The HO storage site was stabilized with Portland cement approximately 30 years ago. Since that time, additional fill materials, including shell, rock, asphalt, and tar, were

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch024

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch024

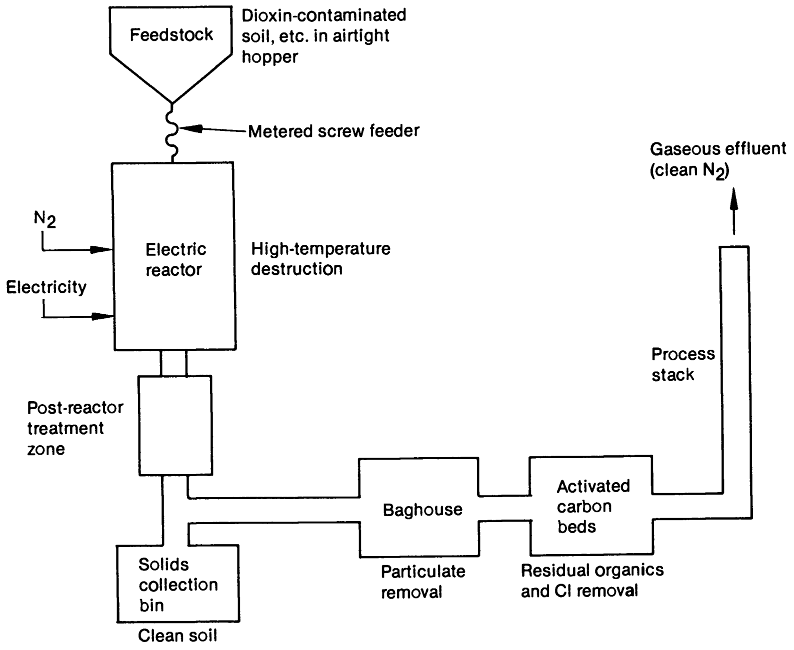


Figure 2. Simplified AER Process Diagram.

added to the storage area, providing a cover ranging up to 10 cm over the cement-stabilized soil.

Before the demonstration testing, a surface and subsurface soil sampling program was conducted to characterize the storage area conditions. At the soil surface, a maximum indicated hot spot concentration of 2,3,7,8-TCDD was found to be 646 ppb. However, the maximum indicated 2,3,7,8-TCDD concentration was 998 ppb, found in the subsurface in the 15-cm-thick cement-stabilized soil. The 2,3,7,8-TCDD concentration in the soil beneath the cement-stabilized soil decreased to a value <1 ppb within a 1-m depth.

Procedure. Testing was performed during June 1985 and included site setup, soil preparation, checkout, treatment of contaminated soil, decontamination, and equipment removal. Soil feedstock was chosen from a plot of known high concentration of 2,3,7,8-TCDD. The material was excavated by use of a shovel, ground to 35 mesh, and loaded into the feed hopper. Analysis of a sample taken from the feedstock showed a 2,3,7,8-TCDD concentration of 111 ppb.

Composition of the feedstock was approximately 50% sand and gravel, 30% shell, 20% cement-stabilized soil, and trace amounts of asphalt and tar. For proper operation of the AER3, it was necessary to pretreat the feedstock soil by reducing the moisture content to less than 1% and crushing the material to pass a 35-mesh sieve with jaw crusher and pulverizer. The feedstock moisture was reduced by placing the material in drums with controlled drum heaters. Comparing sample data before and after this drying process showed some of the TCDD was driven off from the dried soil; however, the effluent was exhausted through a carbon filter to retain released organics.

The NCBC demonstration test, which lasted approximately 58 h, treated 436 kg of soil. The average operating parameters for the run are summarized in Table I. The feed rate normally started from 0.18 kg/min to a constant rate of 0.27 kg/min. For a 2.5-h test, a feed rate of 0.32 kg/min was maintained.

Personnel Protection Equipment. Stringent personnel protection (Class C1) was required whenever soil was being prepared (e.g., grinding and pulverizing) or the AER system was open (e.g., filling feed bins, changing treated soil collection bin, and inspecting the reactor internals). Polyethylene-coated tyvek environmental suits were worn to preclude skin exposure. Soil preparation activities required air-supplied respirators because of dust levels. Full-face respirators were worn for the AER open-system activities. During actual AER operation when the system was closed, less stringent

Table I. AER3 Process Parameters

Parameter	Value
Feed rate	0.25 kg/min
Reactor core temperature	2010°C
Nitrogen flow rate	0.25-0.28 Nm^3/min
Stack gas flow rate	0.377 Nm^3/min
Power requirement	37-40 kW

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch024

personnel protection (Class C2) was used to reduce the chance of heat stress from high ambient temperatures while providing adequate personnel protection.

Process Sampling and Analysis. An independent sampling and analysis program was conducted to verify the detoxification capability of the AER3 operation. Samples of the feedstock, treated soil, baghouse material, and activated carbon filter samples were collected in 470-mL wide-mouth bottles and shipped to an analytical laboratory, which is a certified participant in the EPA Contract Laboratory Program (CLP). A treated soil sample was sent to a second analytical laboratory for comparison of results. Although not a participant in the CLP, this laboratory has performed a variety of special analytical services for EPA, including analysis for dioxins and furans. Analytical methods are given in Table II.

Ambient Air Monitoring. Four high-volume air particulate samplers were operated during field operations for the Huber testing and for another Air Force small-scale technology demonstration being performed at NCBC by the IT Corporation. Sampler 1 was placed upwind for control data. Samplers 2 and 3 were located onsite, just downwind of the operations. Sampler 4 was placed downwind at the perimeter of the storage area to provide exposure data representing the general NCBC workforce and the public. Samplers 1, 3, and 4 were operational whenever there was field activity. Sampler 2 was operated an equivalent amount of time but when there was no field activity to represent an onsite control. The full operation for the two technology demonstrations covered three sampling periods.

Personnel Monitoring. Personnel samples and an area sample were obtained for 2,3,7,8-TCDD analysis. The personnel sampling equipment (mini-ram) was placed on individuals working in the soils preparation room, near the feedstock bin area atop the reactor and the reactor discharge area near the solids collection bin. The area sample was taken inside the trailer near the solids collection bin.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch024

Table II. Analytical Methods

Type	EPA Method
Isomer class dioxins and furans	Method 8280*
2,3,7,8-TCDD	CLP-IFB
Semivolatile organics	CLP-IFB, based on Method 625 (13)
Pesticides and PCB	CLP-IFB, based on Method 608 (13)
Herbicides	Method 8150 (14)
Inorganics	CLP-IFB, based on methods in (15)

* As modified by EPA's Environmental Monitoring and Science Laboratory, Las Vegas. At time of analysis, method was in the review process.

Results and Discussion

The ability of the process to destroy dioxins and furans in the soil is readily seen in Table III, which compares results for the treated soil from the two laboratories with the concentrations in the feedstock. Except for the very low concentration of tetrachlorodibenzofuran (TCDF) (0.028 ppb) found by one laboratory, dioxin and furan isomers were not detectable in the treated soil. The sum of the detection levels for the six isomer classes shows an overall level less than 0.12 ppb and 0.35 ppb for the two laboratories, respectively, which are well within the 1-ppb criteria set for the technology.

The herbicide compounds 2,4-D and 2,4,5-T, found to be 280 and 610 ppm in the feedstock, respectively, were also destroyed during the process to levels less than detectable, which were 0.01 ppm. 2,4,5/2,4,6-trichlorophenol and 2,4-dichlorophenol, found at 55 ppm and 10 ppm in the feedstock, were destroyed to levels less than detectable, which were 1 ppm. Chlorinated organic pesticides and PCBs were not detected in the feedstock; hence, destruction could not be evaluated. Earlier AER tests conducted by Huber on the destruction of PCBs and carbon tetrachloride in soil for Toxic Substance Control Act (TOSCA) and Resource Conservation and Recovery Act (RCRA) permits have shown the process to be highly effective.

Table IV compares the concentrations of analyzed inorganics in the feedstock and treated soil. Zinc and lead were the major elements in the feedstock at 70 and 15 ppm, respectively. Both elements showed significantly reduced concentrations in the treated soil. Lead was reduced the most by 49%. Zinc reduction was 11%. Other detectable elements in the feedstock at low concentrations showed minor gains or losses. Analytical results for the baghouse filtered material show substantial enrichment of zinc and lead (Table IV).

Table III. Dioxin and Furan Concentrations in Feedstock and Treated Soil

	Concentration (ppb)		
		Treated Soil	
Dioxin/Furan	Feedstock	Set A[a]	Set B[b]
Total TCDD	113[c]	<0.036[d]	<0.04
2,3,7,8-TCDD	111	<0.036[d]	<0.04
Total PCDD	3.2	<0.018[d]	<0.03
Total HCDD	1.1	<0.07[d]	<0.02
Total TCDF	12.7	0.028	<0.01
2,3,7,8-TCDF	2.7	0.028	<0.01
Total PCDF	18.8	<0.19[d]	<0.01
Total HCDF	0.65	<0.20[d]	<0.01

a. Analysis by CLP analytical laboratory.
b. Analysis by comparison laboratory.
c. Data taken from 2,3,7,8-TCDD specific analysis.
d. Not detected. Detection limit value shown.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch024

Table IV. Inorganic Concentrations in Feedstock, Treated Soil, and Baghouse Filtered Material

Element	Concentration (ppm)		
	Feedstock	Treated soil	Baghouse
Antimony	<3.[a,b]	<3.[a,b]	<3.[a,b]
Arsenic	5.5	6.4	1.3
Beryllium	<0.3[a]	<0.3[a]	<0.3[a]
Cadmium	<0.3[a]	0.34	3.8
Chromium	6.1	8.2[c]	21.
Copper	6.2	5.8	39.
Lead	15.[b]	7.6	512.
Mercury	<0.1[a]	<0.1[a]	0.4
Nickel	1.6	3.0	9.9
Selenium	<0.5[a,b]	0.58[b]	2.2[b]
Silver	<0.3[a,b]	<0.3[a,b]	<0.3[a,b]
Thallium	<0.5[a,b]	<0.5[a,b]	<0.5[a,b]
Zinc	70.	62.	1260.
Total cyanide	<0.5[a]	<0.5[a]	<0.5[a]

a. Not detected. Detection limit value shown.
b. Spike sample recovery was not within control limits.
c. Anomaly attributed to analysis.

The material enrichment is due to the volatility of these elements within the temperature range of the process. The boiling points for both elements are less than the 2010°C operating temperature. The transport mechanism is likely due to condensation onto an aerosol-sized particulate that was swept to the baghouse filter by the exhaust gas stream. This is analogous to the vapor-condensation phenomenon widely observed on microparticle-sized coal-fired fly ash. Some enrichment is also shown for copper, chromium, and nickel; however, data are insufficient to establish the mechanism. This process shows promise in reducing volatile-type metal contamination in the treated soil and in providing a method of metal recovery from soils heavily contaminated with inorganics.

The treated soil was examined according to EPA's four hazardous characteristics (ignitability, corrosivity, reactivity, and toxicity) to show that the technology produced a residual material that can satisfy the regulation requirements (16) for restoration use. This would permit returning the soil to its original location without further restrictions. The high temperature of the AER process produces glassy spherical particles composed largely of common soil elements. These characteristics ruled out concerns about three of the EPA characteristics: ignitability, corrosivity, and reactivity. The fourth characteristic, extraction procedure (EP) toxicity, was satisfied by showing that the EP extract would never contain amounts of the eight elements, four pesticides, and two herbicides equal to or above the threshold limitations set forth in the regulation (16),

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch024

even if all the amounts could leach from the treated soil. In all cases, there was a factor 10 or more margin between the limits and the individual concentrations.

Ten semivolatile organic compounds were detected in the baghouse material sample at concentrations above 1 ppm, none of which were detected in the feedstock and treated soil samples. The significant compounds and their concentrations were pyrene (47 ppm), acenaphthylene (36 ppm), fluoranthene (33 ppm), naphthalene (18 ppm), and phenanthrene (13 ppm). Their existence is thought to be the result of recombination from destructed components during the pyrolysis process. It is possible during pyrolysis at oxygen deficient conditions, such as in the AER process, to build higher order organic components. The recombination would be occurring near the end of the high temperature process where there is insufficient time for the new organic component to be destroyed. Because these compounds were not detected in the treated soil, the organics must volatize at the high temperature, transport in the exhaust, and later condense on aerosol particles as the exhaust temperature reduces. These results emphasize the need for mitigating features being installed in the exhaust line from the reactor (e.g., baghouse, carbon filters). Because the organic compounds are listed as priority pollutants, the baghouse material can be recycled as feedstock to the reactor to eliminate the hazardous waste.

Problems encountered in the NCBC field demonstration were attributable to pilot-scale operation. Small pieces of metal in the raw feedstock soil caused breakdown of the pulverizer during soil preparation; however, operation of a jaw crusher and screener was within normally expected wear and tear experience. Alignment of the feedstream as it passed through the reactor was a sensitive parameter. At times, melted soil accumulated at the exit of the reactor. The alignment problem has not been experienced in operation of the larger AER12 reactor.

For full-scale operation, Huber has scaled up an AER configuration based on its AER12 experience. Core dimensions are 46 cm in diameter, with a heated length of about 548 cm. A supporting soil pretreatment facility has been designed to blend, dry, pulverize, and size a variety of composite soils of sizes up to 10 in., with an input moisture content up to 30% in the feedstock and an output of 1% moisture content and 35-mesh size. The pretreatment plant and the scaled-up AER are both designed to perform at a soil processing rate of 42 kg/min, with continuous operation seven days a week. The approach is to return the treated soil, as delisted material, to the original excavation area.

Conclusions

The NCBC field test demonstrated that the AER pyrolysis process can meet performance requirements in a reliable manner. Problems encountered were resolved at the HO site and are considered to have no impact on full-scale operation. The six dioxin/furan isomer classes were destroyed in soil to a level of less than 0.12 ppb, which is well within the program objective of 1 ppb. Analytical results show that the treated soil can satisfy EPA requirements for restoration use.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch024

The AER process shows promise in reducing metal contamination in the feedstock. Significant enrichment of lead and zinc in the baghouse filter suggests that the AER process may also be useful in the recovery of inorganics in wastes that can volatilize at AER operating conditions.

Acknowledgments

This activity was conducted by EG&G Idaho, Inc., under a Military Interdependent Purchase Request (FT-8952-85-10009) from the Air Force Engineering and Services Center, Engineering and Services Laboratory, Tyndall Air Force Base, FL, for the U.S. Department of Energy, Idaho Operations Office. The authors wish to thank Mr. D. B. Derrington, Jr., now of Versar Inc., who was the field engineer in charge at the time for the J. M. Huber Corp., and EG&G Idaho chemists W. A. Propp, A. E. Grey, and D. L. Miller for their careful review of the analytical data.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch024

Literature Cited

1. Rappe, C. Environ. Sci. Technol. 1984, 18, 78A-90A.
2. Young, A. L. In "Human and Environmental Risks of Chlorinated Dioxins and Related Compounds"; Tucker, R. E., Young, A. L., Gray, A. P., Eds; Plenum Press: New York, 1981; pp. 173-190.
3. Courtney, K. D.; Gaylor, D. W.; Hogan, M. D.; Falk, H. L.; Bates, R. R.; Mitchell, I. Science 1970, 168, 864-866.
4. Courtney, K. D.; Moore, J. A. Toxicology and Applied Pharmacology 1971, 20, 396-403.
5. Kimbrough, R. D.; Falk, H.; Stehr, P.; Fries, G. J. Toxicol. and Environ. Health, 1984, 14, 47-93.
6. diDomenico, A.; Viviano, G.; Zapponi, G. In "Chlorinated Dioxins and Related Compounds"; Hutzinger, O.; Frei, R. W.; Merian, E.; Pocchiari, F. Eds, Pergamon Press: Oxford, 1982; 105-114.
7. Ahling, B.; Lindskog, A. In "Chlorinated Dioxins and Related Compounds"; Hutzinger, O.; Frei, R. W.; Merian, E.; Pocchiari, F. Eds; Pergamon Press: Oxford, 1982; 215-225.
8. Olie, K.; Lustenhouwer, W. A.; Hutzinger, O. In "Chlorinated Dioxins and Related Compounds"; Hutzinger, O.; Frei, R. W.; Merian, E.; Pocchiari, F. Eds; Pergamon Press: Oxford, 1982; 227-224.
9. Liberti, A.; Brocco, D.; In "Chlorinated Dioxins and Related Compounds"; Hutzinger, O.; Frei, R. W.; Merian, E.; Pocchiari, F. Eds; Pergamon press: Oxford, 1982; 245-251.
10. Choudhry, G. G.; Olie, K.; Hutzinger, O. In "Chlorinated Dioxins and Related Compounds"; Hutzinger, O.; Frei, R. W.; Merian, E.; Pocchiari, F. Eds; Pergamon Press: Oxford, 1982; 275-301.
11. Czuczwa, J. M.; Hites, R. A. Environ. Sci. Technol. 1984, 18, 444-450.
12. Shaub, W. M.; Tsang, W. Environ. Sci. Technol. 1983, 17, 721-730.
13. "Methods of Organic Chemical Analysis of Municipal and Industrial Wastewater", U.S. Environmental Protection Agency, 1982, EPA-600/4-82-057.

14. "Test Methods for Evaluating Solid Waste", U.S. Environmental Protection Agency, 1982, SW-846, Second Edition.
15. "Methods of Chemical Analysis of Water and Wastes", U.S. Environmental Protection Agency, 1979, EPA-600/4-79-020.
16. Title 40, Code of Federal Regulations, Part 261, Identification and Listing of Hazardous Waste, Subpart C, Characteristics of Hazardous Waste.

RECEIVED January 13, 1987

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch024

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch025

Chapter 25

Performance Assessment of a Portable Infrared Incinerator: Thermal Destruction Testing of Dioxin

Philip L. Daily

Shirco Infrared Systems, Inc., 1195 Empire Central, Dallas, TX 75247-4301

The Shirco Portable Unit was built in response to the increasing interest in on-site thermal treatment of waste and successfully demonstrated thermal destruction of dioxin in soil at Times Beach, Mo. The Portable Unit consists of a feed system, a primary chamber fired with electric infrared heating elements, a gas-fired secondary chamber, a wet gas scrubber, monitoring and control systems, and heating element power centers. All equipment is enclosed in a 45 foot trailer. Material is conveyed through the incinerator in a thin sheet on a wire mesh belt, giving all material the same residence time under controlled process conditions. One of the on-site tests performed by the Unit was the thermal destruction of dioxin at Times Beach, Mo. No dioxin was detected in the treated soil, flue gases, or scrubber effluent. The resulting Destruction and Removal Efficiencies (DRE) were greater than 99.9999 % and the particulate emissions were well below 0.08 gr/dscf required by RCRA.

The difficulty of transporting hazardous material to Shirco's Test Facilities in Dallas, Texas, was increased significantly by the implementation of RCRA Regulations. A substantial need existed for a trailer-mounted system which could be moved to waste producing and waste storage sites for thermal treatment testing. Past experience indicated that the ceramic fiber insulated, infrared incinerator was much easier to transport than other similar equipment and was uniquely suited for this type of portable application. In response to this need, Shirco designed and built the Portable Unit to demonstrate the capabilities of a portable infrared incinerator at waste sites. The construction details of the Portable Unit and the results from

0097-6156/87/0338-0311$06.00/0
© 1987 American Chemical Society

the thermal destruction testing of dioxin contaminated soil will be reviewed herein.

Portable Unit Construction

The Portable Unit was designed to demonstrate the performance of the Shirco Infrared Incinerator in many thermal treatment applications. The construction details and process functions of the trailer-mounted incinerator are identical to a full-scale infrared incinerator. The system consists of a feed preparation system, an infrared primary chamber, a gas-fired secondary chamber, a wet gas scrubber, an exhaust system, heating element power centers (HEPC), and data acquisition and control systems. All equipment is enclosed within a 45-foot trailer. A schematic representation of the Portable Unit is shown in Figure 1.

Feed Preparation System. Material for thermal treatment is fed by a pail or inclined conveyor onto a short conveyor located above the primary chamber at the feed end. The feed end is located at the rear door of the trailer to facilitate access. The belt is synchronized with the primary chamber conveyor to control the material feed rate. The drive system includes a clutch for automatic feed shut off should process conditions necessitate. The preparation section includes a hopper mounted over a conveyor belt. The conveyor is shrouded and has rubber skirts to minimize air leaking in or furnace gases escaping out of the incinerator. An adjustable knife gate at the conveyor discharge distributes the material across the width of the belt. Final sealing in the feed area is provided by an additional adjustable knife gate in the feed chute at the entrance to the primary chamber.

Primary Chamber. The primary chamber has a rectangular cross-section and has an external shell of carbon steel with nominal dimensions of 2.5 ft wide X 9 ft long X 3 ft high. The installed weight of the chamber is 3000 pounds. The inner chamber is lined with multiple layers of ceramic fiber blanket insulation. The insulation is mounted on stainless steel studs and retained by ceramic fasteners. The feed material is conveyed through the incinerator on a wire mesh belt which rides upon high-temperature alloy shafts supported by externally mounted bearings. The belt is woven with alloy steel wire. A friction drive system pulls the belt through the primary chamber. When the material reaches the discharge end, it drops from the belt into an enclosed hopper. The hopper contains a sampling drawer for collection of ash samples during operation. A discharge screw conveyor is located at the bottom of the tapered hopper and transports the treated material out of the trailer into a sealed collection drum. The primary chamber top is constructed such that the gas flow within the chamber can be either counter-current or co-current. Infrared energy is supplied by heating elements which are transversely mounted across, and equally spaced along, the length of the chamber. The elements are silicon carbide rods and are connected in wireways external to the chamber. The connections are accessed by

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch025

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch025

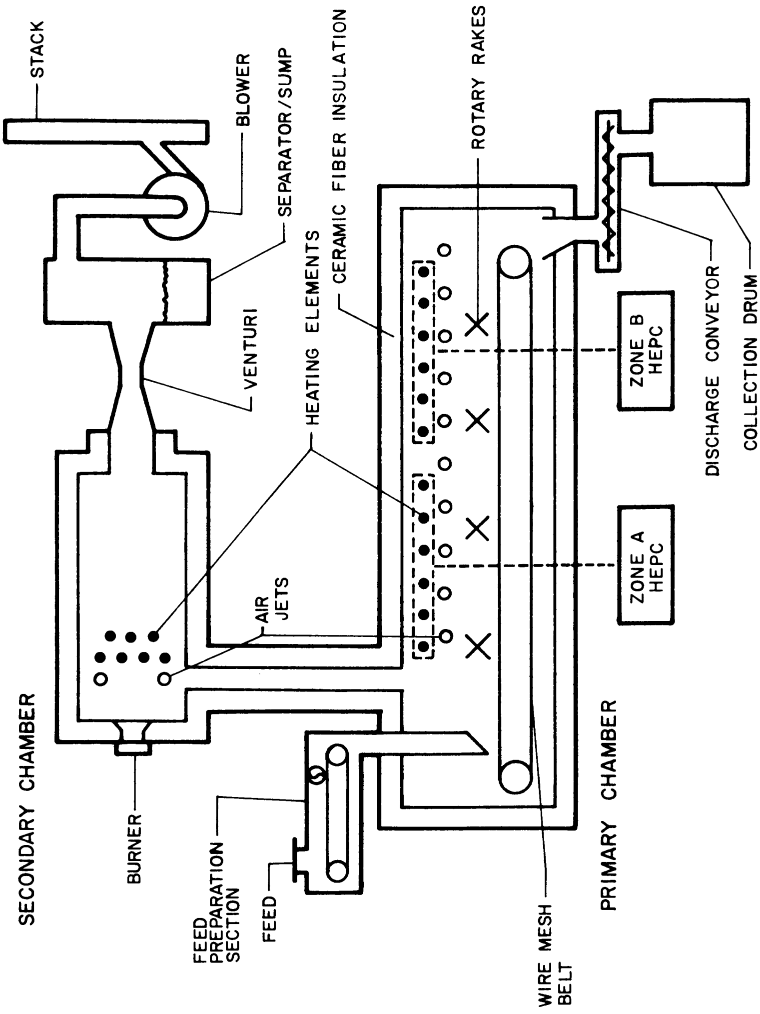


Figure 1. Shirco portable unit schematic.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch025

removing wireway covers. The heating elements are grouped into two control zones. Each zone is powered by a 12 KVA heating element power center (HEPC). The processing capabilities include up to an 1850°F temperature, with the residence time of solid phase material variable between 10 and 180 minutes. Any atmosphere, oxidizing, reducing, or neutral, can be used. An exhaust duct which is internally insulated with ceramic fiber insulation connects the primary chamber and secondary chamber.

Secondary Chamber. The secondary chamber has a rectangular cross-section with a carbon steel shell with external dimensions of 3 ft wide X 9 ft long X 3 ft high. The chamber weighs 1,500 pounds and is lined with ceramic fiber blanket insulation. A propane-fired burner ignites any combustible gases carried over from the primary chamber and provides the energy to maintain the primary chamber exhaust gases at a predetermined temperature. An array of silicon carbide bars are installed to provide turbulence for good gas mixing. The chamber is sized to provide sufficient gas residence time for combustion of the gases (typically 1.5 to 2.5 seconds). The process temperature can be maintained at up to 2300°F with a 2.2 second gas residence time and up to 100 % excess air. The burner is mounted in the chamber end plate, with the flame pattern intersecting the incoming primary chamber exhaust gases at a 90° angle as they enter the secondary chamber. Electronic flame monitoring is included with the burner for automatic shutdown upon loss of flame. Provisions were made in the chamber end plate on either side of the burner to add injection of liquids.

Combustion Air. Combustion air for the primary and secondary chambers is supplied by a blower which manually adjusts the air flowrate. A splitter manifold with two dampers is located at the blower outlet and provides control of airflow to both chambers. Air is injected at 10 points on either side and along the length of the primary chamber through a manifold system. Adjustment of the flow to the injection tubes is by a gate valve. In the secondary chamber, air is injected through two jets on either side of the chamber, directed at the intersection of the burner flame and incoming gas flow. The jets are offset to produce a swirling action of the gases. The air flowrate is adjusted by means of a gate valve and the burner air registers.

Wet Gas Scrubber. Exhaust gases from the secondary chamber pass through a wet gas scrubber. This system consists of a venturi section containing liquid sprays and a separator tower with additional liquid sprays and a sump tank. A cone-type damper is located in the venturi section and is used to control scrubber pressure drop. The sprays in the venturi inject a fine liquid mist into the exhaust stream to agglomerate particulate grains. The larger and heavier particulate grains are then removed in the separator tower by gravity, centrifugal force, and the additional liquid sprays. The scrubber also cools the gases from their incoming temperature (1000°F to 2300°F depending on processing conditions) to saturation temperature, usually about 180°F. Subcooling can be performed, but consumes substantially

more liquid. A recirculation pump is used to transport the liquid from the sump tank to the scrubber sprays. Reactive material, such as lime or caustic soda, can be added to the scrubber sump tank for acid removal.

Exhaust System. An induced draft exhaust blower is located on the discharge side of the scrubber. The blower is capable of exhausting the primary and secondary chambers, producing a slight draft on the system, while overcoming the pressure drop of the scrubber. A butterfly damper is located on the outlet side of the blower to provide a means of adjusting the exhaust gas flow. A removable exhaust stack is installed through the trailer roof. The stack extends 10 feet above the trailer roof and is equipped with two standard EPA sampling ports. Access to the sampling ports is provided by scaffolding installed beside the trailer.

Data Acquisition and Control. The system includes a master control cabinet which contains process controllers, alarm and status lights, and data recording and monitoring equipment. The cabinet has a temperature controller for each of the two primary chamber heated zones. A six point temperature recorder and a twelve point thermocouple switch with digital indicator are used for temperature monitoring and recording. Table I lists the process instrumentation included for monitoring and control of the Unit. The main electrical components use lights to annunciate their status. Alarm lights are also installed in the cabinet to annunciate high primary chamber temperature, an open wireway cover, both low and high secondary chamber temperature, and high stack temperature.

Table I. Portable Unit Process Instrumentation

Measured Variable	Identification
Temperature	Primary Chamber (5 points)
	Primary Chamber Exhaust
	Secondary Chamber
	Secondary Chamber Exhaust
	Stack
	Discharge Chute
Pressure	Primary Chamber
	Secondary Chamber
	Burner Gas
	Combustion Air
	Venturi Water
	Separator Tower
Level	Scrubber Water
Flowrate	Venturi Sprays
	Separator Tower Sprays
Time	Primary Chamber Run Time
Voltage	all 3 phases, each HEPC
Current	all 3 phases, each HEPC
Power	each HEPC

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch025

Heating Element Power Center (HEPC). The heating elements in the two primary chamber zones are powered by two heating element power centers. The primary power connection is to a 3-phase, 480-volt source. Each HEPC has a transformer and a power control unit. The power control unit (PCU) regulates the power by the phase-angle firing method. A bank of silicon control rectifiers (SCR) adjusts the current waveform, allowing only a percentage of the incoming power to reach the transformer section. The transformer receives power from the PCU and lowers the voltage, increasing the current, of the heating element circuit. The transformer and PCU are mounted in a ventilated enclosure.

Thermal Destruction Testing

Times Beach, Missouri, had a population of over 2000 when the roads were sprayed with waste oil for dust control. The roads were later found to be contaminated with dioxin. The property in the town was eventually purchased by the U.S. Government and is now deserted and under guard with restricted entry.

Shirco talked with the Missouri Department of Natural Resources in early 1985 about the Times Beach site and convinced them of the Unit's capability based on these discussions and their observation of another test. Subsequently, the decision was made to take the Unit to the Times Beach site to demonstrate its capability with dioxin decontamination.

The Portable Unit arrived at the Times Beach Dioxin Research Facility on July 8, 1985, to demonstrate decontamination of the dioxin laden soil. The Unit was set up and made ready for operation within a few hours after arrival to the site. This rapid setup demonstrated its unique capability for mobility. The testing personnel followed a safety plan developed by the Missouri DNR which included pretest physical examinations, use of Class C safety clothing, and vital sign monitoring and rest periods for personnel. The weather was hot and humid which required the work in the trailer to be limited to 20 minutes at a time.

The soil used for testing was contaminated with 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) at a concentration of about 200 ppb. Two tests were run at a 48 lb/hr feed rate and solid phase residence times of 30 minutes and 15 minutes. The primary chamber temperature was 1490°F to 1560°F. The secondary chamber temperature was maintained above 2200°F. The thermal treatment conditions for both tests are listed in Table II. The testing was performed over two days followed by decontamination of the incinerator and trailer. The incinerator was decontaminated by an extended, high temperature bake out.

Flue gas samples were taken over a 7 hour period for the 30 minutes residence time case and over a 2.5 hour period for the 15 minute case. These sampling times were chosen so that sufficient gas sample could be collected to demonstrate the required destruction efficiency. The flue gas sampling was conducted in accordance with a modified EPA Method 5 procedure and the ASME dioxin protocol (1). The samples were collected isokinetically at a single stack sampling point. At the conclu-

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch025

sion of each test run, the sampling train was taken to a designated clean area for sample recovery. Orsat analyses of the flue gases were also performed using EPA Method 3. Several grab samples were collected of the thermally treated soil for each run condition. Samples from the scrubber recirculation system were collected to provide a composite, 1000 ml sample for each run. All samples were placed in a precleaned, 1000 ml amber glass container for subsequent analysis. Laboratory analyses for the TCDD isomer were performed using a gas chromatograph coupled with tandem mass spectrometry (GC/MS/MS).

Table II. Incinerator Operating Conditions

	Solid Phase Residence Time (minutes)	Feed Rate (lb/hr)	Primary Chamber Zone A Temp. (°F)	Primary Chamber Zone B Temp. (°F)	Secondary Chamber Temp. (°F)
Test #1	30	47.68	1560	1550	2250
Test #2	15	48.12	1490	1490	2235

The sampling results are presented in Table III and indicate that the flue gas, the treated soil, and the scrubber effluent were free of dioxin at the detection limits listed. The particulate sampling results indicate that the particulate was a small percentage of the 0.08 gr/dscf RCRA Regulation requirement.

The flue gas and feed material sampling data were combined for the calculation of the destruction and removal efficiencies. Table IV tabulates the calculation of the DREs for both

Table III. Results of Dioxin Destruction Testing

Sample Stream	Units	Test #1	Test #2
Analyses for 2,3,7,8-TCDD:			
Waste Feed	ng/g	227	156
Treated Soil*	ppt	<38	<33
Scrubber Effluent*	ug/l	-	<1
Flue Gas*	pg	<14	<8.4
Analyses for Particulate:			
Filter Catch at 7 % O_2	gr/dscf	0.0010	0.0002
Orsat Analyses of Flue Gas:			
CO_2	%	3.8	3.0
O_2	%	14.4	10.0
CO	%	0.0	0.0
N_2	%	81.8	87.0

* TCDD was non-detectable and shown at detection limit

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch025

test runs. These calculations show that the DREs for 2,3,7,8-TCDD were in excess of the EPA criteria of 99.9999 %. These results indicate that the infrared incinerator can very effectively decontaminate dioxin contaminated soil while maintaining compliance with all state and federal emissions requirements.

Table IV. Calculation of Destruction and Removal Efficiency for 2,3,7,8-TCDD

	Units	Test #1	Test #2
Waste Feed Inlet:			
Waste Feed Rate	kg/hr	21.68	21.83
TCDD Concentration	ug/kg	227	156
TCDD Mass Rate	ug/hr	4921	3405
Exhaust Gas Outlet:			
Sample Volume	dsm^3	7.066	3.044
Stack Gas Flowrate	dsm^3/hr	97.19	129.99
TCDD in Sample	pg	<14	<8.4
TCDD Concentration	pg/m^3	<1.981	<2.760
TCDD Mass Rate	pg/hr	<192.6	<358.7
Destruction and Removal Efficiency (DRE)	%	>99.999996	>99.999989

Conclusion

The Portable Unit has successfully demonstrated its capability for thermal treatment of hazardous wastes at the source of the material. This type of on-site treatment would eliminate the need of transportation of hazardous materials to a distant site of stationary treatment equipment. The Portable Unit also has demonstrated that it can be moved to a site and be ready to treat material very quickly, a capability which will be very important in operation of full scale equipment. The on-site treatment of the Times Beach dioxin contaminated soil resulted in no dioxin detected in any of the incinerator effluent streams. The product of the testing activity was soil with no detectable level of dioxin. Dioxin contaminated soil thermally treated in this manner will yield soil which can be disposed as non-hazardous material. The decontamination was performed without exceeding RCRA requirements for particulate emissions and with dioxin destruction efficiencies surpassing the required percentage. The overall conclusion was that the infrared incinerator can very effectively remove dioxin from contaminated soil at the source of the contaminated soil.

Literature Cited

1. "Sampling For the Determination of Chlorinated Organic Compounds in Stack Emissions," ASME Draft, October, 1984.

RECEIVED December 15, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch025

Chapter 26

Technology Demonstration of a Thermal Desorption–UV Photolysis Process for Decontaminating Soils Containing Herbicide Orange

R. Helsel[1], E. Alperin[1], T. Geisler[1], A. Groen[1], R. Fox[1], T. L. Stoddart[2], and H. D. Williams[3]

[1]International Technology Corporation, 312 Directors Drive, Knoxville, TN 37923
[2]Headquarters, Air Force Engineering Services Center, Research & Development Directorate, Environics Division, Environmental Engineering Branch, Tyndall Air Force Base, FL 32403
[3]Waste Technology Programs, EG&G Idaho, Idaho Falls, ID 83415

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch026

Laboratory and field testing determined the effectiveness of a new decontamination process for soils containing 2,4-D/2,4,5-T and traces of dioxin. The process employs three primary operations - thermal desorption to volatilize the contaminants, condensation and absorption of the contaminants in a solvent, and photochemical decomposition of the contaminants. Bench-scale experiments established the relationship between desorption conditions (time and temperature) and treatment efficiency. Laboratory tests using a batch photochemical reactor defined the kinetics of 2,3,7,8-TCDD disappearance. A pilot-scale system was assembled to process up to 100 pounds per hour of soil. Tests were conducted at two sites to evaluate treatment performance and develop scale-up information. Soil was successfully decontaminated to less than 1 ng/g 2,3,7,8-TCDD at temperatures above 460°C.

As part of a major program being conducted by the U.S. Air Force to restore to normal use several Department of Defense sites where soils have been contaminated with low levels of Herbicide Orange (HO), International Technology Corporation (IT), under subcontract to EG&G Idaho, has been conducting a project involving laboratory bench-scale and field pilot-scale tests to demonstrate a new soil treatment process - thermal desorption/UV photolysis (TD/UV). The intent of the demonstration was to reduce the combined tetra-, penta-, and hexa-chlorinated dibenzodioxin (CDD) and furan (CDF) congeners, which originated from the HO, to less than 1 ng/g, which represented the anticipated soil clean-up criteria. Treatment should also effectively remove the primary HO constituents, 2,4-D

0097-6156/87/0338-0319$06.00/0
© 1987 American Chemical Society

and 2,4,5-T. Two sites were included in the field demonstration project for the TD/UV process, each having substantially different types of soil but reasonably similar concentrations of the HO constituents. Testing at the Naval Construction Battallion Center (NCBC) at Gulfport, Mississippi was conducted by IT during May 1985; testing at Johnston Island (JI) in the Pacific Ocean occurred in July 1986. Based on the results of these field pilot demonstrations, an engineering and cost evaluation is being performed for applying TD/UV technology using large, mobile systems for these two sites or other sites having similar contaminated soil problems. This paper describes the technology, highlights the results of the initial laboratory test phase, and summarizes the field demonstration results.

Process Description

The thermal desorption/UV photolysis process developed by IT accomplishes substantial volume reduction and toxicity reduction by concentrating the hazardous constituents contained in the soil into a small volume which is easier to treat than large quantities of soil. The process incorporates three steps:

- Desorption - heating the soil to volatilize the organic contaminants
- Scrubbing - collecting the volatilized organics in a suitable solvent
- Photolysis - converting the contaminants to relatively non-hazardous residues through photochemical reactions.

A schematic block-flow diagram is presented as Figure 1. Contaminated soil is passed continuously through an indirectly heated desorber which can be one of many types of conventional equipment applicable for thermal processing of solids. The treatment performance of the desorber is controlled by the residence time and temperature of the soil. Treatment requirements (i.e., operating conditions) are determined by the volatility of the soil contaminants and the required contaminant removal efficiency (final versus initial concentration).

The off-gas leaving the desorber contains organic vapors, water vapor originating as initial soil moisture, and small quantities of air which enter with the soil. Scrubbing using a high boiling hydrocarbon solvent is used to treat the off-gas to remove the organic contaminants and water vapor by cooling, condensation, and absorption. Particulates (e.g., fine soil) which may be entrained by the off-gas are also collected by the scrubbing solvent. Scrubbed off-gas is passed through a conventional emission control system, such as carbon adsorption, to ensure that no organic contaminants or solvent vapors are released. Scrubber solvent is recirculated to the scrubber after being processed through a system of phase separation, filtration, and cooling. Condensed water, which is immiscible with the solvent, is separated and either directly treated using conventional techniques, such as filtration and carbon adsorption, or discharged to an existing

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch026

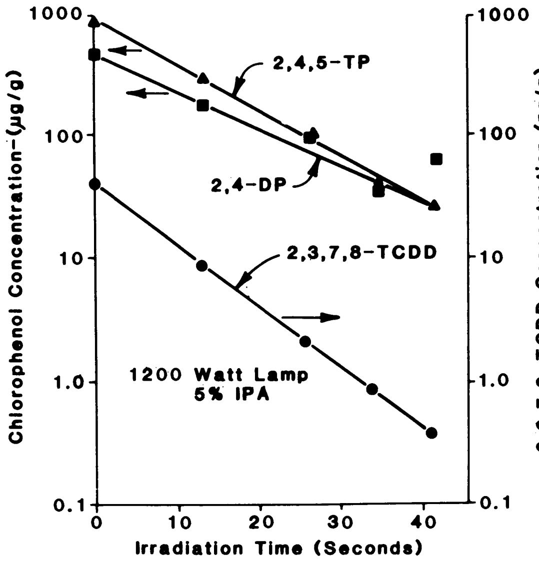


Figure 1. Thermal desorption/UV photolysis process concept.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch026

wastewater treatment facility. Filtered solids are recycled to the desorber or packaged as process waste for off-site disposal, depending on the relative quantity and composition.

A small portion of the recirculated solvent stream is diverted to a UV photolysis system to treat (detoxify) and remove the organic contaminants, with the treated solvent purge recycled to the scrubbing system. The equilibrium concentration of the contaminants in the scrubber solvent is maintained as high as practical to minimize the purge stream and afford higher photolysis reaction rates, thereby decreasing the size of the photolysis treatment system. The concentration limitation is dependent on the solubility properties and partial pressure of the contaminants in the solvent, and the resultant effect on scrubber efficiency and emission potential. The photolysis system contains a specially designed flow reactor which subjects the contaminant-laden solvent to UV radiation to induce molecular decomposition. High intensity mercury vapor lamps produce a band of wavelengths, some of which match the absorption energy of the specific organic molecules being treated. Cooling is provided to the reactor to remove the thermal output of the lamp. The photolyzed solvent is treated by using selected conventional physical or thermal separation processes, such as distillation, to remove the reaction product residue. Alternatively, a purge of the photolyzed solvent can be discarded as waste to control the levels of reaction products in the recirculated solvent system.

Other configurations of treatment processes using thermal desorption as the primary separation technique can be applied to organically contaminated soils. Alternative physical/chemical processes can be used to treat the desorber off-gas and the contaminants. To achieve complete contaminant destruction, the off-gas can be treated by using conventional fume incineration or other thermal treatment technology. The choice of the type of desorber and off-gas treatment system depends on the concentration and properties of the chemical contaminants, soil characteristics, quantity of contaminated material, site characteristics, availability of off-site disposal, and regulatory and related requirements.

Laboratory Testing and Results - Thermal Desorption

Thermal desorption is a physical separation process, although chemical transformation of the organic contaminants may occur depending on the thermal stability and the operating temperatures required to achieve adequate decontamination efficiency. Thermal desorption has been used only in a limited number of cases (1-4) for treating contaminated soil, and these applications have involved relatively volatile organic compounds, such as solvents. Because of the extremely low volatility of CCD and CDFs, the development of basic treatability data was essential to confirm that 1 ng/g levels in soil could be achieved and that the required desorption conditions were practical, considering the design features and operating rates of equipment available for performing such treatment.

Desorption treatability testing was conducted on samples of contaminated soil from three HO contaminated sites - NCBC, JI, and

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch026

Eglin Air Force Base. The goals of the test effort were to evaluate the effect of time and temperature on 2,3,7,8-TCDD removal efficiency and to establish the importance of soil type. The samples were selected by the Air Force based on results of site surveys to yield high contamination levels in order to investigate a broad range of treatability. This testing was an extension of earlier testing performed for the EPA on two dioxin-contaminated soil samples from Missouri to support EPA's mobile incinerator trial burn in 1985 (5).

After each soil sample was blended, air dried, and screened (2 mm sieve opening) triplicate aliquots were taken and analyzed for 2,3,7,8-TCDD, CDD and CDF congeners, and 2,4-D and 2,4,5-T. The three prepared soils had 2,3,7,8-TCDD levels greater than 100 ng/g and 2,4-D/2,4,5-T levels of about 1000 µg/g. The JI soil had significant concentrations of hepta and octa CDD compared with the other two samples. In addition, selected physical and chemical properties presented in Table I, were measured (6). The EPA test program (5) had indicated that soil properties had only a minor influence on removal efficiencies for 2,3,7,8-TCDD.

Table I. Physical-Chemical Analysis of Prepared Soil Samples Used for Laboratory Thermal Desorption Tests

Parameter	JI	Eglin	NCBC
pH	8.4	3.8	8.6
Conductivity (millimhos/cm)	5.0	0.15	0.21
Organic matter (percent)	4.2	1.2	2.3
Cation exchange capacity milliequivalents/100g)	0.73	0.77	2.4
Oil and grease content (grams/100g)	0.19	0.41	0.34
Surface area (m^2/g)	6.7	2.5	12.3
Particle size distribution (percent)			
Medium sand	41	41	26
Fine sand	37	52	59
Silt	19	5	12
Clay (<5 microns)	3	2	3
Moisture	2.3	0.79	1.1

A series of 10 individual tests was performed using temperatures between 430 and 560°C and treatment times of 8 to 30 minutes. Table II presents the test results, which are comparable to the earlier results for Missouri soils. The objective of 1 ng/g 2,3,7,8-TCDD residual in soil was achieved for all three soils subjected to the highest temperature. There was some difference in treatability observed between the three soils at the lower temperatures. Also, longer treatment times were required for the NCBC soil because of the higher initial 2,3,7,8-TCDD level (500 ng/g vs. 100 ng/g). One set of treated test samples which contained less than 1 ng/g 2,3,7,8-TCDD was also analyzed for the other CDD and CDF congeners and 2,4-D/2,4,5-T. These results, shown in Table III, indicate greater than 99.999 percent removal of the initial

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch026

2,4-D/2,4,5-T and the effective removal of higher chlorinated CDDs and CDFs.

Table II. Effect of Treatment Conditions on Residual 2,3,7,8-TCDD in Soil During Laboratory Thermal Desorption Tests

Nominal Test Temperature (°C)	Soil Identification	Time at Test Temperature (min)	2,3,7,8-TCDD Concentration (ng/g)	
			Initial	Final
430	JI	20	106	38.5
	Eglin	20	101	4.4
	NCBC	30	494	26.6[a]
481	JI	15	106	4.5
	JI	30	106	1.6
	Eglin	15	101	1.1[a]
	Eglin	30	101	0.45
	NCBC	15	494	10.1
	NCBC	30	494	4.6
558	JI	8	106	0.56[a]
	Eglin	8	101	0.71
	NCBC	15	494	0.76[a]

[a]Average of duplicate tests or duplicate analyses.

Table III. Residual 2,4-D, 2,4,5-T, and CDD/CDF in Soil Samples Treated at 558°C in Laboratory Thermal Desorption Tests

Compound	Concentration (ng/g) JI	Eglin	NCBC
2,4-D	ND[a]	ND	ND
2,4,5-T	16	0.8	3
TCDF	0.6	0.4	ND
OCDF[b]	0.3	ND	ND

[a]ND = Not detected.
[b]No other CDD and CDF congeners were detected.

Laboratory Testing and Results - Photolysis

Photolysis has had limited application for treatment of hazardous waste or detoxification of chemically contaminated materials. The susceptibility of chlorinated aromatics, including herbicides such as 2,4-D and 2,4,5-T, to UV-induced decomposition is well established (7,8). Photodecomposition of such compounds leads to successive dechlorination followed by condensation reactions to form phenolic polymers (7,8). Other research has demonstrated that CDD and CDF decompose in the presence of UV light (8,9,10). Development of a photochemical process for destroying 2,3,7,8-TCDD in a waste tar indicated similar dechlorination and condensation reactions and products (8). The high-molecular weight end products, which are similar in structure to humic acids, would be expected to have low toxicity and mobility. Therefore, essentially complete

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch026

conversion of toxic constituents could produce a potentially non-hazardous (according to RCRA), easily disposable residue.

Laboratory photolysis experiments were designed to confirm that 2,3,7,8-TCDD contained in the selected scrubber solvent could be reduced to 1 ng/g and to determine the reaction rates of the primary HO constituents and 2,3,7,8-TCDD in that solvent matrix. A previous photolysis process for 2,3,7,8-TCDD used hexane as a solvent (8). The solvent selected for use in the TD/UV process was different - a high boiling (kerosene-like) mixture of isoparaffins. This hydrocarbon solvent was selected because of its very low vapor pressure and water solubility, nontoxic and nonflammable characteristics, relatively low cost, chemical stability, and good solvent properties for HO constituents. A second major difference from earlier IT photolysis studies was the presence in the scrubber solution of significant concentrations of other chlorinated organic reactants (2,4-D and 2,4,5-T) which were also subject to photolysis. In fact, the typical concentration ratio between 2,4-D or 2,4,5-T and 2,3,7,8-TCDD in the soil samples used in the desorption treatability testing was 2000:1.

The three steps of the laboratory program included generation of scrubber solution, bench-scale batch photolysis reactions, and a pilot system trial. In order to generate a representative sample of scrubber solution for photolysis tests, a small desorption and scrubbing system was assembled. A portion of the prepared samples of both NCBC and JI soil used for the thermal desorption tests was used to generate scrubber solution. Contaminated soil (~100 g) was placed in a standard tube furnace apparatus which was heated to about 500°C for 15 to 30 minutes. A nitrogen purge swept the vapors into the scrubbing system, which consisted of several solvent-filled impingers. Analysis of the prepared scrubber solutions indicated thermochemical conversion of the 2,4-D and 2,4,5-T in the contaminated soil to the corresponding chlorophenols at molar equivalents. In addition to using prepared scrubber solutions, solvent spiked with 2,4-D and 2,4,5-T, the corresponding chlorophenols, or 2,3,7,8-TCDD was used for baseline photolysis tests.

Most photolysis experiments were conducted in a 0.5 liter capacity standard quartz photochemical reactor using either recirculation or bottom agitation for heat and mass transfer. Both 100- and 450-watt high pressure quartz mercury vapor lamps (Canrad-Hanovia, Inc., Catalog Nos. 608A and 679A) were used, depending on the initial reactant concentration in the particular solvent solution being tested. The wavelengths of interest based on spectrophotometric absorbance measurements of 2,3,7,8-TCDD, 2,4-D and 2,4,5-T were in the 280 to 320 nm region. Isopropyl alcohol (~0.05 g/g solvent solution) was used as a proton donor to minimize formation of polymeric reaction by-products which tend to foul the light transmission surfaces (8). The bench-scale photolysis tests gave the following results:

1. All compounds disappeared to below the analytical detection limits.
2. The concentration of 2,3,7,8-TCDD was reduced to less than 1 ng/g from initial concentrations as high as 200 ng/g.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch026

3. For a given reactor configuration and lamp wattage, the reaction rates of 2,3,7,8-TCDD and 2,4,5-trichlorophenol were proportional to the concentration, indicating pseudo-first order kinetics in agreement with previous work (8).
4. Absorbence of UV energy by the solvent, which increased during irradiation, resulted in low quantum yields and low rate constants.
5. Insoluble brown reaction products (presumably phenolic tars) were deposited on the surfaces of the reactor vessel and lamp well. This expected phenomenon plus the high solvent absorbence demanded a careful reactor selection and photolysis system design.

Trials using a pilot reactor system described in the following section were performed in the laboratory to establish reactor efficiencies and operating characteristics prior to transport to the field. A synthetic scrubber solution was prepared containing 2,4,5-trichlorophenol at a concentration (~2,000 μg/g) projected to be representative of the planned field tests. Kinetics were determined to be first-order with a rate constant of 0.07 sec^{-1}

On-site Pilot Testing and Results

Based on the information developed from the laboratory test program, a pilot-scale TD/UV system was designed and assembled. Three skids were used to mount the desorber, scrubber, and photolysis systems; the largest skid was 1.5 meters by 4.3 meters. A conventional pilot-scale, rotary, indirect-fired calciner was used as the desorber. The calciner consisted of a 3.3 meter long by 16 cm internal diameter rotating tube through which the soil was transferred, and a gas-fired furnace which surrounded the middle 2.0 meters of the tube length. The initial and final tube sections were used for soil feeding and cooling. The flow rate and residence time of soils traveling through the desorber were controlled by varying the tube inclination and rotational speed. Temperature of the soil was measured at different locations by a thermowell probe extending inside the tube. Soil was fed to the desorber from a small hopper using a variable speed screw conveyor. Soil leaving the tube was collected in a sealed metal can.

The off-gas transfer and scrubbing system was designed to enable recirculation of scrubbed off-gas through the desorber. The entire off-gas treatment and recirculation system, including the desorber and scrubber, was operated at a slightly negative pressure to prevent potential fugitive emissions. A small amount of air entered the system with the soil feed or through seal leakage. Nitrogen was added to the recirculated gas stream to maintain the oxygen concentration below the level necessary to support combustion. This was an extra safety feature since the vapor pressure of the solvent at normal scrubber operating conditions is very low. A portion of the scrubbed off-gas was vented from the recirculation system to maintain proper pressure in the system. This purge stream was passed through a small HEPA filter and carbon adsorber before being discharged to the atmosphere. The solvent system

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch026

consisted of a scrubber, receiving and separation tank, storage tank, recirculation pump, filters for removing suspended solids, and solvent cooler.

The photolysis system was independent of the desorber and scrubber systems; its design capacity was lower than necessary to match the desorber's soil-processing rate. A portion (about 40 kg) of contaminant-laden solvent was taken from the scrubber system after completion of one or more desorption tests and transferred to the photolysis system. This system consisted of an agitated storage tank, solvent recirculation pump, and photochemical reactor with associated cooling, DC power supply, and controls. The selected type reactor was a standard quartz falling-film unit, approximately 10 cm in diameter and 50 cm long (Ace Glass, Inc., Part No. 7898). A 1200 watt high intensity mercury vapor lamp was inserted through a central quartz tube within the reactor to irradiate the solvent as it flowed by gravity down the circumference of the reactor body. The solvent was recirculated through the reactor for many cycles to achieve sufficient irradiation (e.g., reaction) time.

Five desorption tests were carried out at NCBC at various treatment conditions. A total of 800 kg of soil was processed; soil was prepared by drying and crushing to less than 1/2 inch to allow proper flow in the desorber feed mechanism, and blending for uniformity. Each test lasted 5 to 10 hours, including the heat-up and cool-down cycle. Samples of feed soil and treated soil were taken during steady-state operation, and samples of the scrubber solvent and vent carbon were taken at the conclusion of each run. Samples were analyzed for 2,4-D, 2,4,5-T, other HO indigenous compounds, priority pollutant organics and metals, and tetra-hexa congeners of CDD and CDF. In addition, 2,3,7,8-TCDD concentrations of treated soil and photolyzed solvent samples were determined on a quick-response basis to enable adjustment of the operating conditions in subsequent tests. Fresh solvent and carbon were used for each test, and the entire desorber and scrubber network was cleaned out between tests. This cleaning enabled thorough inspection of the condition of the equipment and provided several different compositions of contaminated solvent to use in the photolysis tests.

Table IV shows the effect of different soil temperatures and residence times on residual 2,3,7,8-TCDD for NCBC pilot tests. Table V presents the analytical results for 2,4-D, 2,4,5-T, and total CDD and CDF. Analytical detection levels for 2,3,7,8-TCDD and the various congeners were generally less than 0.1 ng/g but varied from sample to sample, ranging from 0.018 ng/g to 0.51 ng/g.

All test conditions produced soil containing less than 1 ng/g 2,3,7,8-TCDD. The total quantified tetra-hexa congeners were less than the treatment goal of 1 ng/g for the first three tests, which were performed at the lower feed rates. Test 4, made at the highest feed rate, nearly met this value, whereas the much lower soil temperature used for the final test resulted in almost 3 ng/g combined residual CDD and CDF. A longer residence time could have improved this performance. Residual 2,4-D and 2,4,5-T concentrations were less than 1 μg/g for all but the final test. This reduction represents greater than 99.97 percent removal efficiency for these primary HO constituents.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch026

Table IV. Effect of Treatment Conditions on Residual 2,3,7,8-TCDD During NCBC Pilot Thermal Desorption Tests

Test No.	Soil Feed Rate (kg/hr)	Residence Time[a] (min)	Soil Temperature (°C)	2,3,7,8-TCDD (ng/g) Initial	Residual
1	13.6	40	560	260	ND
2	13.6	40	560	272	ND
3	25	19	560	236	ND
4	44	10.5	560	266	ND
5	20	24	460	233	0.5

[a]Soil residence time in heated zone.

Table V. Residual 2,4-D, 2,4,5-T, and CDD/CDF in NCBC Pilot Thermal Desorption Test

Compound	Concentration (ng/g) Test 1	Test 2	Test 3	Test 4	Test 5
2,4-D	180	150	20	-	170
2,4,5-T	500	270	60	-	1240
TCDD	ND[a]	0.23	0.11	0.61	0.75
PCDD	ND	ND	ND	ND	ND
HCDD	ND	ND	ND	ND	ND
TCDF	ND	ND	ND	0.13	0.95
PCDF	ND	0.14	ND	0.54	1.0
HCDF	ND	ND	ND	ND	ND
CDD and CDF[b]	ND	0.37	0.11	1.28	2.70

[a]ND = not detected.
[b]Total of quantified values for detected cogeners.

Because of the very low moisture content of the prepared soil feed, an insufficient volume of aqueous condensate was collected from the tests to perform analysis or treatability tests. A vent gas sample was taken, but no valid analytical results were generated because of delays in sample processing. However, analysis of the carbon used in the emission control adsorbers enabled some evaluation of scrubber performance and process emission potential. Only the front (upstream) portion of carbon from one of the tests showed detectable levels of any CDD or CDF. No HO constituents were detected in the downstream portion of carbon. Calculated scrubber removal efficiencies exceeded 99.9 percent for CDD, CDF, 2,4-D, and 2,4,5-T. Vent gas volume was about 0.05 m^3/minute for all tests.

Results of the photolysis tests are presented in Table VI. The total solvent volume (~10.5 l) was recirculated through the reactor at 0.75 l/min for 6.5 hr, resulting in 28 cycles with an irradiation time of about 1.5 sec/cycle. The photolysis system operating time was selected based on the laboratory trials to achieve less than 1 ng/g 2,3,7,8-TCDD; the actual residual level of

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch026

0.36 ng/g represented greater than 99 percent conversion. The reaction conversion of the other CDD and CDF congeners varied from 85 to 99 percent. Photolysis reduced the concentrations of 2,4-dichlorophenol (2,4-DP) and 2,4,5-trichlorophenol (2,4,5-DP), (corresponding to the 2,4-D and 2,4,5-T present in the initial soil) by 85 and 97 percent respectively. Figure 2 shows the rate of disappearance of 2,3,7,8-TCDD, 2,4-DP, and 2,4,5-TP. As demonstrated during the laboratory tests, the reaction kinetics were pseudo-first order over the given range of concentrations. The reaction rate constants were similar for the three species (0.11 sec^{-1}, 0.04 sec^{-1}, and 0.08 sec^{-1}, respectively); the rate constant for 2,4,5-TP was comparable to that determined in the laboratory trials of the pilot system.

Table VI. Initial vs Final Concentration of Selected Compounds in Scrubber Solution from NCBC Pilot Photolysis Tests

	Concentration (ng/g)	
Compound	Initial	Final
2,4-Dichlorophenol	490,000	82,000
2,4,5-Trichlorolorophenol	977,000	31,000
2,3,7,8-TCDD	43.3	0.36
Total TCDD	46.3	0.92
Total PCDD	15.7	2.3
Total HCDD	0.84	0.037
Total TCDF	31.0	3.8
Total PCDF	3.7	1.1
Total HCDF	1.7	0.0031

Three desorption tests and one photolysis test were conducted at JI to compare the effects of different soil characteristics and investigate higher processing rates. The coral-like soil used for the tests contained lower levels of HO contamination than NCBC (about 50 ng/g versus 250 ng/g). As much as 95 kg/hr of soil was successfully decontaminated to less than 1 ng/g 2,3,7,8-TCDD using desorption temperatures of 550°C. Treated soil from all three desorption tests had nondetectable residual tetra-hexa CDD and CDF cogeners, 2,4-D and 2,4,5-T, and corresponding chlorophenols. Analysis of carbon removed from the desorber-scrubber system vent showed no detectable concentration of CDD or CDF. Gas samples taken downstream of the carbon adsorber showed nondetectable concentrations of CDD and CDF, 2,4-D and 2,4,5-T, and chlorophenols. Photolysis test results were comparable with NCBC tests. Initial concentrations of HO contaminants were much higher in the scrubber solvent due to processing of considerably more soil and use of less solvent. The concentration of 2,3,7,8-TCDD was reduced from 780 ng/g to less than 0.7 ng/g during 12 hours of system operation (representing about 80 sec reaction or irradiation time). Total chlorophenols were reduced from 430 µg/g to less than 6 µg/g, and tetra-hexa CDD and CDF cogeners were effectively treated. Reaction rate constants for specific compounds were essentially the same between the NCBC and JI photolysis tests. At JI as at NCBC, brown residues were deposited on the reactor surfaces, and solvent discoloration was obvious, but there was no evidence of rate retardation.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch026

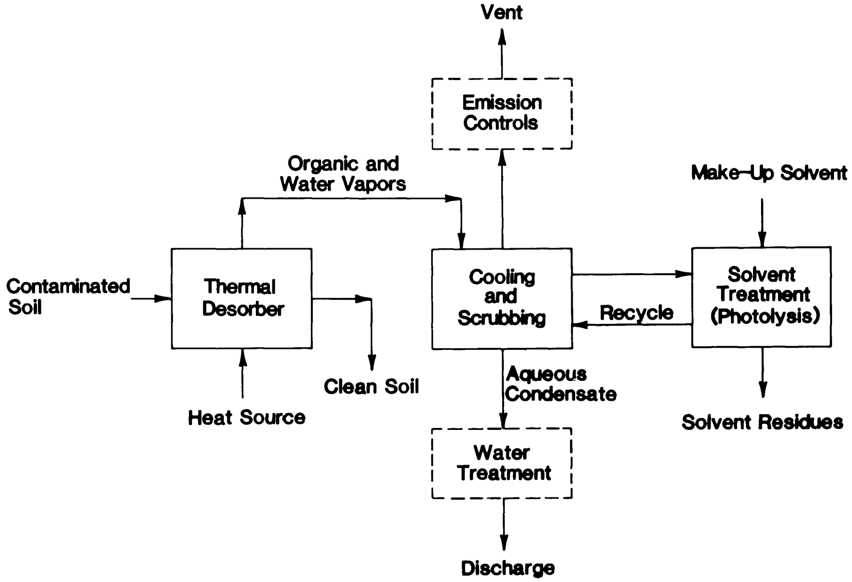


Figure 2. Photolysis reaction kinetics for pilot test at NCBC.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch026

Conclusions

The effectiveness of thermal desorption to decontaminate soil containing HO and of UV photolysis to destroy HO toxic constituents has been demonstrated in bench- and pilot-scale tests. Some additional technical information is needed for a complete evaluation of the process and to provide the basis for design of a full-scale system for on-site remedial action. This project illustrates the requirements for developing and implementing new process technology for solving contaminated-soil environmental problems. Only through such demonstration efforts can more cost-effective and environmentally sound remedial action alternatives be made available.

Literature Cited

1. Noland, J. W.; NcDevitt, N. P.; Koltuniak, D. L. Proc. of the National Conference on Hazardous Wastes and Hazardous Materials, Atlanta, GA, March 4-6, 1986, pp. 229-232.
2. Hazaga, D; Fields, S; Clemmons, G. P. The 5th National Conference on Management of Uncontrolled Hazardous Waste Sites, Washington, DC, November 7-9, 1984, pp 404-406.
3. Webster, David M. J. Air Pollution Control Association, 1986, 36, pp 1156-1164.
4. Hoogendoorn, D. Proc. of the 5th National Conference on Management of Uncontrolled Hazardous Waste Sites, Washington, DC, November 7-9, 1984, pp 569-575.
5. Helsel, R.; Alperin, E.; Groen, A.; and Catalano, D. "Laboratory Investigation of Thermal Treatment of Soil Contaminated With 2,3,7,8-TCDD," draft report to U.S. EPA, Cincinnati, OH on Work Order BAD001, D.U.D-109, IT Corporation, Knoxville, TN, Dec. 1984.
6. Arthur, M. F.; Zwick, T. C. "Physical-Chemical Characterization of Soils," Battelle Columbus Laboratories, Columbus, OH, 1984.
7. "Report on 2,4,5-T, A Report on the Panel on Herbicides of the President's Science Advisory Committee," Executive Office of the President, Office of Sciences and Technology, March 1971.
8. Exner, J. H.; Johnson, J. D.; Ivins, O. D.; Wass, M.N.; and Miller, R. A. "Detoxication of Hazardous Waste," Ann Arbor Science Publishers, Ann Arbor, MI, 1982, p 269.
9. Exner, J. H.; Alperin, E. S.; Groen, A.; Morren, C. E.; Kalcevic, V.; Cudahy, J. J.; and Pitts, D. M. "Chlorinated Dioxins and Dibenzofurans in the Total Environment," Keith, L. H.; Rappe, C.; Choudhary, G.; Eds., Butterworth Publishers, Stoneham, MA, 1985, p 47.
10. Exner, J. H., Alperin, E. S.; Groen, A; Morren, C. E. Hazardous Waste, 1, 1984, pp 217-223.

RECEIVED December 29, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch026

Chapter 27

In Situ Radio Frequency Heating Process for Decontamination of Soil

H. Dev[1], P. Condorelli[1], J. Bridges[1], C. Rogers[2], and D. Downey[3]

[1]IIT Research Institute, Chicago, IL 60616
[2]U.S. Environmental Protection Agency, Cincinnati, OH 45268
[3]Headquarters, Air Force Engineering Services Center, Tyndall Air Force Base, FL 32403

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch027

In-situ radio frequency (RF) heating is accomplished by inserting tubular electrodes into boreholes, or by laying horizontal electrodes over the surface of the soil, and connecting them to a source of electromagnetic (EM) energy in the frequency range of 6 to 13 MHz. An overview of the RF heating techniques is presented. Experiments were performed on 75 g batches of sandy soil to determine the feasibility of removing chlorinated hydrocarbons. Tetrachloroethylene was selected as a simulant. Soil containing 9.28 and 957 ppm tetrachloroethylene was heated for four hours in a temperature range of 90° and 130°C. It was demonstrated that under these conditions 95% of the contaminant can be removed from soil.

Radio Frequency (RF) in-situ heating is an electromagnetic (EM) technique originally developed and demonstrated by Krstansky et al. (1) for in-situ thermal processing of hydrocarbonaceous earth formations for resource recovery. This technique uses EM energy in the radio frequency band for rapid in-situ heating of earth and mineral formations. The mechanism of absorption and conversion of EM energy to heat is similar to that of a microwave oven, except that the frequency is lower and the scale of operation is much larger. In-situ RF heating to 200°-400°C of large blocks of earth from 35 cu ft (1.0 cu m) to 660 cu ft (25 cu m) in size has been demonstrated in field tests (1). A sustained average heating rate of 0.8°-1.0°C/hr was achieved in these tests.

The purpose of this paper is to present the results of laboratory studies performed to determine the feasibility of using RF heating techniques to decontaminate soils containing hazardous chemicals such as chlorinated hydrocarbons, benzene, and toluene. The occurrence of numerous large uncontrolled sites of contaminated soil containing the above-mentioned chemicals is well documented and previously reported in the literature (2-6).

In-situ RF heating offers two alternatives for decontaminating soil. These alternatives are: (1) thermal decontamination of soil

0097-6156/87/0338-0332$06.00/0
© 1987 American Chemical Society

by vaporization and recovery of contaminants, and (2) in-situ RF heat treatment in conjunction with the application of a chemical dechlorinating agent for contaminants such as PCB. In alternative (1) the contaminants will be vaporized, distilled, or steam-stripped from the soil and recovered at the surface. In alternative (2) RF heating of the soil will be used to improve the rate of reaction between the contaminant and the applied reagent. In addition, the heating can be used to condition the soil by removing the moisture either before or after reagent application.

Thermal Decontamination of Soil

Chlorinated organic contaminants are found at various sites of interest to the U.S. Air Force. Among these contaminants are compounds such as tetrachloroethylene, dichloroethane, trichloroethylene, chlorobenzene, benzene, toluene, and components of JP-4 jet fuel. These materials have a boiling range of 80° to 232°C, have substantial vapor-pressure at 100°C, and can be steam distilled if present in excess of their solubility limit. To establish the feasibility of thermal recovery of such chemical contaminants, tetrachloroethylene (120.8°C, nbp) was selected as a representative contaminant. Uncontaminated (clean) sandy soil from the vicinity of a waste site was spiked with tetrachloroethylene and used in recovery experiments.

Experimental

Procedure. A large amount of sandy soil was obtained from the field. Approximately 1000 g of this soil was placed in a large glass jar and tumbled overnight. A soil sample was analyzed for chlorinated solvents by extraction in a Nielson-Kryger (7) steam distillation apparatus followed by analysis of the extract on a gas chromatograph (GC) equipped with an electron capture (EC) detector. When it was verified that the soil did not contain any chlorinated solvents, the moisture content of the soil was adjusted to 5 or 10%.

The clean moist soil was prepared for experiments by placing it in the 500 ml round bottom flask and spiking it with a solution of tetrachloroethylene to a concentration of either 9.28 or 957.3 ppm. The flask was sealed and rotated in an ice bath for 3 hr to homogenize the soil and the spike by tumbling. At the end of this tumbling period, the flask was attached to a water-cooled solvent-recovery condenser. The side leg of the condenser was placed in a chilled receiver containing approximately 10 ml of pesticide grade hexane. A Tenax trap was placed on the gas outlet port of the condenser to trap uncondensed vapors.

The soil was heated with a heating mantle to the desired final temperature and maintained at that temperature for 4 hr. At the end of this time, the heating mantle was removed and the flask was cooled to room temperature. The condenser was washed with hydrocarbon-free water and hexane. These washings were combined with the distillate. The Tenax trap was also washed with pesticide grade hexane and the washings were combined with the distillate. The distillate and washings were dried by passing through a bed of anhydrous sodium sulfate. The dried distillate and washings were

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch027

brought up to volume with hexane in a 100 ml volumetric flask and analyzed for tetrachloroethylene on a Hewlett Packard 5840 GC equipped with a Ni^{63} EC detector.

The glass flask containing the treated soil was attached to the Nielson-Kryger distillation head to determine the residual tetrachloroethylene. The amount of tetrachloroethylene extracted by this procedure was corrected for the extraction efficiency. The extraction efficiency of tetrachloroethylene from sandy soil was determined by extraction of soil spiked at nine levels in the concentration range of 0.1 to 1190 ppm. The average extraction efficiency based on all nine samples was 97.1% with a standard deviation of ±4.3.

Results. The results of decontamination experiments are summarized in Tables I and II. The results of all high concentration experiments (Table I) show that the average recovery was 96.6% with a standard deviation of ±2.0. The results were not affected significantly by varying the temperature in the range of 90° to 130°C. Similarly, recovery was not affected by varying soil moisture in the range of 5.7 to 9.85 wt%. Similar results were obtained when soil containing 9.28 ppm tetrachloroethylene and approximately 5.7% moisture was treated at 90° and 100°C (Table II). The average recovery of tetrachloroethylene in five experiments using 9.28 ppm tetrachloroethylene and approximately 5.7% moisture at 90° and 100°C was 95% with a standard deviation of ±1.5.

The results show that substantial recovery of tetrachloroethylene from soil is feasible in the temperature range of 90° to 130°C. Considering the data shown in Tables I and II, and extrapolating the result obtained at a concentration of 957.3 ppm heated to 130°C, it may also be stated that there is no significant difference between the amount of tetrachloroethylene recovered when the soil is heated to 90° and when it is heated to 130°C.

Table I. Thermal Recovery of Tetrachloroethylene from Soil (containing 957.3 ppm)

Initial Moisture, %	Temp., °C	Time, hr	Residual Concentration, ppm	Recovery, %
5.9	21	4.00	1089‡	0.4
5.7	90	4.00	58.4	94.2
5.7	90	4.10	52.0	94.9
5.7	101	3.55	29.1	97.1
5.7	101	3.62	18.4	98.2
5.7	101	4.02	56.3	94.5
5.7	131	3.75	20.6	98.0
9.85	89	4.00	1.6	99.8
9.85	102	3.50	25.0	97.6

‡Initial concentration: 992 ppm. Difference is within the bounds of experimental error.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch027

Table II. Thermal Recovery of Tetrachloroethylene from Soil (containing 9.28 ppm)

Initial Moisture, %	Temp., °C	Time, hr	Residual Concentration, ppb	Recovery, %
5.9	21	4.0	9690‡	0.3
5.7	89	4.07	242	97.5
5.7	90	3.73	499	94.9
5.7	99	3.58	461	95.3
5.7	100	3.72	635	93.5
5.7	101	3.45	525	94.7

‡Initial concentration: 9.92 ppm. Difference is within the bounds of experimental error.

Principles of Radio Frequency Heating

The term radio frequency (RF) generally refers to the frequencies used in wireless communications. These frequencies can be as low as 45 Hz or extend well above 10 GHz. The frequencies primarily used for radio frequency, dielectric, or microwave heating range from 6.78 MHz to 2.45 GHz.

The principles of RF heating are similar to those of a microwave oven, except that the frequency of operation is different and the size of the application is much larger. In these systems, the temperature rise occurs as a result of ohmic or dielectric heating mechanisms. Ohmic heating arises from an ionic current or conduction current that flows in the material in response to the applied electric field. Dielectric heating results from the physical distortion of the atomic or molecular structure of polar materials in response to an applied electric field. Since the applied AC electric field changes rapidly, the alternating physical distortion dissipates mechanical energy that is translated into thermal energy in the material.

The dielectric properties of soil determine the amount of RF power that can be dissipated in the soil. These properties are the relative dielectric constant (ε_r) and the loss-tangent. The loss-tangent, tan δ, is defined as $\sigma/\omega\varepsilon_o\varepsilon_r$ where σ is the apparent conductivity, ω is the frequency of the applied electric field, radians/sec, and ε_o is the permittivity of free space which equals 8.85×10^{-12} Farads/meter. All the dielectric properties are a function of soil temperature, the frequency of the applied field, and the composition of the soil.

The amount of RF power dissipated in the soil is directly related to the frequency of the applied electric field, to the square of the amplitude, to the relative dielectric constant, and to the loss-tangent (8).

The depth of penetration of the electromagnetic energy is measured by its skin depth. Skin depth is defined as the distance from the power source at which the amplitude of the EM wave falls to

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch027

37% of its initial amplitude (8). Skin depth is inversely related to the square root of frequency, apparent AC conductivity, or the loss-tangent.

The dielectric parameters determine the depth of penetration of EM fields into the soil for a given operating frequency. If the depth of penetration is small compared to that needed, only the volume nearest the energy source would be heated initially. This suggests that for any reasonable volume, as low a frequency as possible should be chosen. On the other hand, if too low a frequency is chosen, very little energy absorption will occur for an acceptable level of electric field. While the electric field can be increased, electrical breakdown, corona discharge, or other undesirable effects can result. Thus, there is an optimum range wherein a suitable frequency can be chosen for a given volume of material and a given set of dielectric parameters.

In-Situ RF Heating Systems

A fully operational in-situ RF heating system for decontamination requires the development and testing of at least four major sub-systems. These are: (1) RF energy deposition electrode array; (2) RF power generation, transmission, monitoring and control system; (3) vapor barrier and containment system; and (4) gas and liquid condensate handling and treatment system.

Among the sub-systems mentioned above, the design of the electrode array (also called the exciter array) is the critical factor that will determine the design requirements and constraints for the other three sub-systems.

Previous attempts (9-12) to use electrical energy for heating earth formations were aimed at resource recovery from hydrocarbonaceous deposits. Simple techniques such as burying electrical heating elements or a pair of electrodes to which 60 cycles AC power is applied were not successful for two main reasons: (1) nonuniform heating led to unacceptable levels of energy inefficiency; and (2) heating beyond the boiling point of free water was not possible. To overcome the temperature limitation imposed by 60 Hz heating, antennas radiating very high frequency or microwaves have been considered (9). Though these methods provide rapid volumetric heating even above the boiling point of free moisture, they suffer from inefficient use of the applied energy.

To overcome these limitations, it is necessary to use bound-wave exciters as opposed to the radiated wave horns or antennas previously used. The bound-wave exciters are designed to fully contain the EM radiation within a defined volume of soil. There are two basic types of bound-wave exciter arrays. These are the tri-plate line and the fringing-field transmission line.

Triplate Line. The triplate transmission line is the rectangular analogue of the more familiar cylindrical coaxial cables. The triplate line is formed by a fully enclosed rectangular cavity in which a central planar conductor has been inserted parallel to the large sides of the cavity. Clearly emplacement of solid metal plates that enclose a rectangular cavity below the soil surface is impractical. This problem has been resolved (1, 13) by simulating the fully contained rectangular cavity by inserting an array of

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch027

electrodes into bore-holes drilled through the soil. The electrodes are inserted in three parallel rows that represent the two outer walls and the central conductor of the fully contained rectangular triplate. It has been demonstrated (1, 13) that through appropriate selection of the row-spacing and the spacing of the electrodes within each row, it is possible to fully contain the applied electromagnetic field within the two outer rows of electrodes.

The triplate line is suitable for those soil decontamination applications where the contaminants have penetrated more than three feet below the surface and for contaminants that require treatment temperatures in excess of 130° to 150°C.

Figure 1 illustrates a conceptual design of such an array that is covered by a vapor barrier to contain and collect gases and vapors as they rise from the surface of the soil. A triplate line was tested by Krstansky et al. (1) by heating 660 cu ft of a tar sand formation to 200°C to an average depth of 14.7 ft.

Fringing-Field Line. This concept makes use of the fringing fields or leakage fields that exist near a transmission line. Figure 2 illustrates a schematic diagram showing rows of horizontal electrodes placed over the surface of the soil. Fringing fields are formed around the electrodes when they are energized. Thus EM energy is first absorbed by the moist layers of soil nearest the electrodes. As the moisture is driven out of the soil, the energy is selectively absorbed at greater depths. The heating zone is eventually restricted because of the exponential fall-off of the fields into the soil.

A 1:3 scale model of a single electrode from the fringing-field line shown in Figure 2 was made and tested (14). The results of this test show that the fringing field line is suitable for the treatment of soils in which the depth of contaminant penetration is less than three feet and where the required treatment temperatures do not exceed 130°C. Additional development and testing is, however, necessary to fully explore the limitations of the fringing field lines, and in particular, to determine ways to improve the penetration depth.

Cost Estimate

Preliminary cost estimates for treating contaminated soil using the tri-plate exciter have been reported (14). It was estimated that the cost of treating soil containing 12.1% moisture to a temperature of 170°C was $42/ton. This estimate was based on the treatment of a 3-acre site to a depth of 8 ft by the progressive heating of a 96 x 96 x 8 ft module. It was estimated that the 3-acre site can be treated in one year. Appropriate pilot and field tests currently in the planning phase will validate operating and economic parameters.

Conclusions

Laboratory-scale studies have shown that 95% of the tetrachloroethylene present in soil can be recovered by heating to a temperature range of 90° to 130°C for 4-hr. Additional experiments are necessary to determine whether similar recovery efficiencies can be sustained over deep soil beds. The RF heating techniques have

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch027

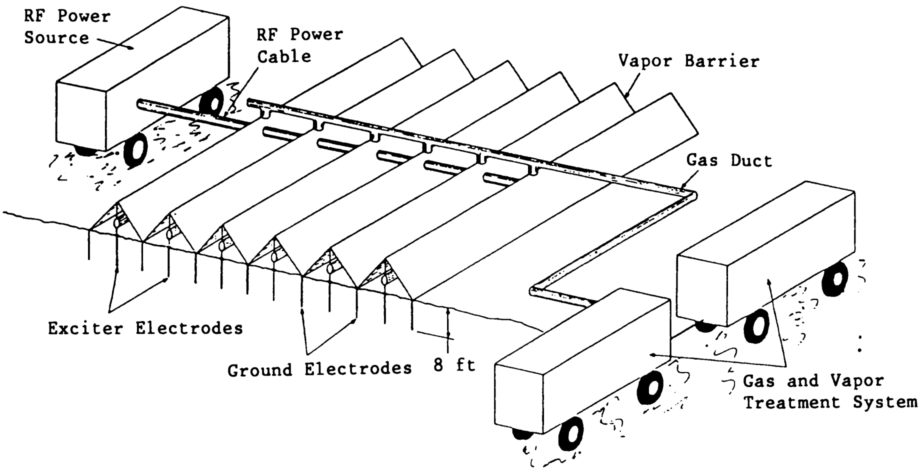


Figure 1. In situ treatment module for area 10,000 ft^2, depth 8 ft.

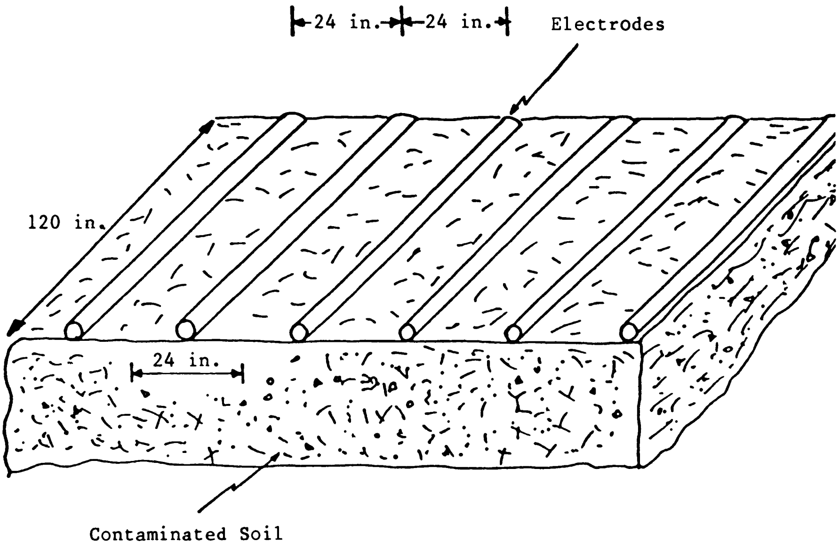


Figure 2. Schematic representation of a fringing field line (size 10 x 10 ft).

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch027

been tested on hydrocarbonaceous earth formations in the temperature range of 200 ^{o}C to 400 ^{o}C. Appropriate field and pilot tests are necessary to validate the technical feasibility and the cost parameters of using the in situ RF heating methods for soil decontamination.

Acknowledgment

This work was supported by the U.S. EPA, USAF, and IIT Research Institute through a U.S. EPA co-operative agreement CR-811529-01-0.

Literature Cited

1. Krstansky, J., et al. "RF Heating of Utah Tar Sands"; IIT Research Institute: Chicago, 1982; Final Report, IITRI E06482.
2. Barnhart, Benjamin J. Environmental Science Technology 1979, 12, 1132-6.
3. Murray, C. Chemical & Engineering News 1979, 57, 12-16.
4. Rogers, C. J. Proc. 8th Annual EPA Symposium on the Treatment of Hazardous Waste, 1983, p. 197.
5. "Solid Waste Facts - A Statistical Handbook," U.S. Environmental Protection Agency, Office of Public Awareness, 1978.
6. "Everybody's Problem: Hazardous Waste," U.S. Environmental Protection Agency, Office of Water and Waste Management, 1980.
7. Onuska, F. I; Terry, K. A. Analytical Chemistry 1985, 57, 801-5.
8. Dev, H., J. Bridges, and Clark, D. "Radio Frequency Enhanced Decontamination of Soils Contaminated with Halogenated Hydrocarbons, Interim Report"; IIT Research Institute; Chicago, 1985; IITRI C06600.
9. Abernathy, E. R. 1974 Annual Technical Meeting of the Petroleum Society of CIM, Calgary, Alberta, 1974, paper 374007.
10. Flock, D. L; Tharin, A. J. Journal of Canadian Petroleum Technology 1975, 14, 17.
11. Ljungstrom, F. Teknisk Tidskrift 1951, 81, 33.
12. Salomonsson, G. Tidskrift För Tekniski-Vetenskaplig För Skning 1953, 24, 118.
13. Sresty, G. C.; Dev, H.; Snow, R. H.; Bridges, J. E. SPE Reservoir Engineering 1986, I, 85.
14. Dev, H. Proc. 12th Annual Research Symposium, 1986, p. 402. EPA/600/9-86/022.

RECEIVED December 5, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch027

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch028

Chapter 28

Biodegradation of Chlorinated Organic Compounds by *Phanerochaete chrysosporium*, a Wood-Rotting Fungus

John A. Bumpus and Steven D. Aust

Department of Biochemistry, Michigan State University, East Lansing, MI 48824

The white rot fungus, Phanerochaete chrysosporium, is able to mineralize a number of environmentally persistent organochlorides such as 1,1-Bis(4-chlorophenyl)2,2,2-trichloroethane (DDT), polychlorinated biphenyls (PCBs), 2,3,7,8-tetrachlorodibenzo[p]dioxin (2,3,7,8-TCDD), Lindane (1,2,3,4,5,6-hexachlorocyclohexane), and pentachlorophenol (PCP). Studies suggest that the ability to degrade these compounds is dependent upon the lignin degrading system of this fungus. For example, we have shown that, like ^{14}C-lignin mineralization, mineralization of ^{14}C-pentachlorophenol (^{14}C-PCP) and ^{14}C-DDT is promoted in nutrient nitrogen deficient cultures of P. chrysosporium whereas their mineralization is suppressed in nutrient nitrogen sufficient cultures. Also, the temporal onset and disappearance of both ^{14}C-PCP and ^{14}C-DDT mineralization appeared similar to that observed for ^{14}C-lignin, thus suggesting that the same general degradative system may be responsible. It is suggested that the ability of P. chrysosporium to mineralize such a wide variety of organochlorides may make this fungus a useful microorganism for use in the biological treatment of contaminated soils, sediments and aqueous wastes when used in appropriate aerated waste treatment systems.

During the past 40 years large quantities of chlorinated organic compounds have been manufactured for use worldwide. Some of these compounds, such as the pesticides DDT, Lindane and pentachlorophenol, were purposely introduced into the environment to control various noxious or harmful plant and animal pests (1). Others, such as polychlorinated biphenyls, contaminated the environment through use or as waste from manufacturing processes (1). Some, such as 2,3,7,8-TCDD, were unknowingly introduced as contaminants of other compounds or preparations (1).

0097-6156/87/0338-0340$06.00/0
© 1987 American Chemical Society

Regardless of the manner by which they were introduced into the environment, organohalides as a group represent an environmentally persistent class of chemicals, many of which accumulate in the food chain in the body fat of animals occupying higher trophic levels (1). Many of these compounds are toxic, mutagenic and/or carcinogenic. Furthermore, many sites exist worldwide that are contaminated with these compounds. Often these sites are old production or use facilities which contain high concentrations of the pollutant in question and represent potential health hazards. Of great concern in many cases is the threat of ground water contamination. Thus these sites are candidates for decontamination and reclamation.

Recent attention has focused on the possible usefulness of the white rot fungus Phanerochaete chrysosporium for the biodegradation of hazardous and environmentally persistent organohalides (2-7). This paper summarizes our present knowledge concerning the ability of this fungus to degrade halogenated organic compounds.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch028

Phanerochaete chrysosporium and the Biodegradation of Lignin. Lignin is an abundant, naturally occurring polymer whose function in nature is to provide structural support to woody plants (8). Its formation in vivo is catalyzed by the free radical oxidative polymerization of cinnamyl alcohols (8). Because the type and quantity of cinnamyl alcohols may vary and because its biosynthesis occurs via a non-stereospecific free radical mechanism, the lignin polymer is a racemic heteropolymer whose structure varies from species to species (8). The lack of an ordered and repeating structure coupled with the racemic nature of the polymer (8) combine to make lignin resistant to attack by most enzyme systems.

Initial studies in our laboratory (9) and by others (10-15) suggested that, like lignin biosynthesis, lignin biodegradation by the white rot fungus P. chrysosporium also proceeded via a free radical process. It had been known for some time that hydrogen peroxide (H_2O_2) is secreted by many wood rotting fungi (16,17) and it was suspected that H_2O_2 was required for lignin biodegradation (17). Forney et al. (9) subsequently presented evidence which showed that, in cultures of P. chrysosporium, H_2O_2 gave rise to the formation of hydroxyl free radicals (Figure 1), a very reactive oxygen species that is able to cause depolymerization of the lignin polymer (13). Furthermore, it was shown that H_2O_2 formation coincided with the onset of lignin mineralization in P. chrysosporium (Figure 2) (9). Subsequent work in a number of laboratories (18-25) has confirmed the requirement for H_2O_2 and that an active oxygen species is indeed required for lignin depolymerization and/or degradation. However, these detailed studies of lignin degradation in P. chrysosporium have shown that the most important oxygen activation step is enzyme-mediated by a family of H_2O_2-requiring hemeproteins, collectively referred to as ligninases, that are secreted by this fungus during idiophasic metabolism in response to nutrient starvation (18-25). By catalyzing various oxidations of the lignin molecule these enzymes mediate cleavage of many C-C and C-O bonds, thus achieving depolymerization. Furthermore, Schoemaker et al. suggest that carbon centered free radicals of lignin-derived metabolites may be able to diffuse away from the

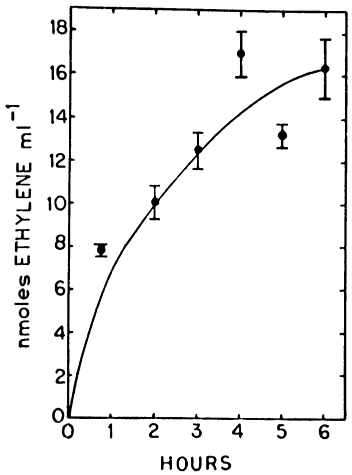


Figure 1. The production of hydroxyl radical (•OH) in ligninolytic cultures of P. chrysosporium as detected by ethylene formation. The •OH was detected by the •OH-dependent formation of ethylene following the addition of α-keto-γ-methiolbutyric acid (KTBA) (3.3 mM final concentration) to cultures grown for 14 days in nutrient nitrogen deficient media. The ethylene produced was detected using gas chromatography and is expressed as nanomoles of ethylene/ml of headspace. Reproduced with permission from Ref. 9.

ligninases and catalyze further oxidation of lignin at sites that are remote from the active site of the enzyme (26). Once the initial oxidative depolymerization of lignin occurs, the smaller and more soluble lignin derived metabolites then undergo further modification and, ultimately, metabolism to carbon dioxide.

Although the most important factor in the initial oxidative depolymerization of lignin is undoubtedly the catalytic action attributed to the ligninases, the hydroxyl radical may still play a role in lignin degradation (26). Additionally, a manganese dependent peroxidase, which is also secreted by this fungus may have an important function in lignin degradation (24,27-29). Regardless of the precise mechanism of lignin degradation, it is apparent that this system is a very non-specific and non-stereoselective biodegradation system. It is this lack of specificity that makes this fungus a potentially useful microorganism for the treatment of hazard-

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch028

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch028

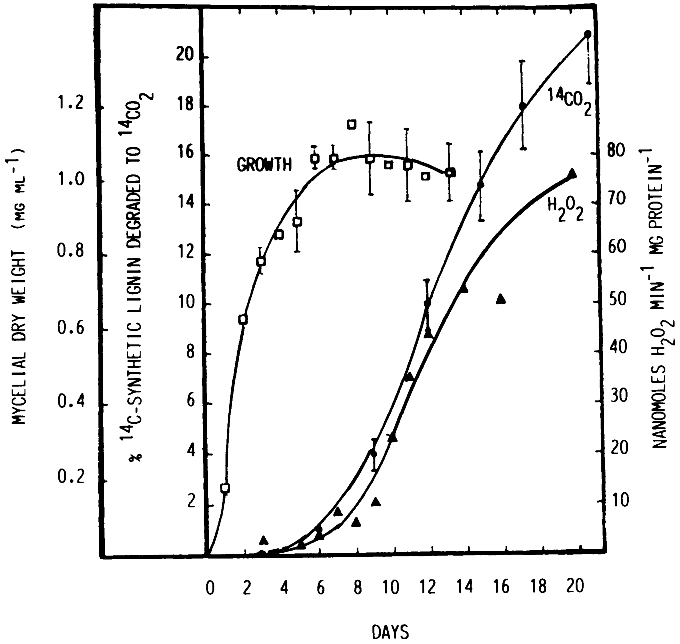


Figure 2. Relationship between the specific activity for H_2O_2 production in cell extracts (Δ), the metabolism of 2' ^{14}C-labeled synthetic lignin to $^{14}CO_2$ (O), and mycelial dry weight (□). P. chrysosporium was grown in low nitrogen medium. Reproduced with permission from Ref. 9.

ous chlorinated organics. Furthermore, a free radical mechanism would allow cleavage of C-Cl bonds.

Degradation of Chlorinated Compounds by P. chrysosporium: An Overview. The first use of P. chrysosporium in the treatment of chlorinated organic compounds focused on the treatment of chlorinated lignin (30) and chlorinated lignin-derived low molecular weight compounds that are by-products of the bleaching procedure that is often used with the Kraft pulping process (6,7). In these studies, it was demonstrated that even highly chlorinated compounds like tetrachloroguiacol could be degraded by the lignin degrading system of this organism (6,7). Other studies showed that chlorinated lignins, like their nonchlorinated precursors, could be degraded to carbon dioxide (30). A somewhat surprising finding was that the chlorinated lignin was mineralized faster than unmodified lignin (30).

The fact that P. chrysosporium degraded chlorinated lignin and lignin by-products, coupled with the finding that lignin degradation proceeded via a non-specific free radical process, suggested to us that even more recalcitrant organohalides might be degraded by this microorganism (2-4). We subsequently demonstrated that a number of normally persistent organohalides (DDT, Lindane, 2,3,7,8-TCDD, 3,4,3'4'-tetrachlorobiphenyl (3,4,3',4'-TCB), 2,4,5,2',4',5'-hexachlorobiphenyl (2,4,5,2',4',5'-HCB)) as well as the polyaromatic hydrocarbon benzo[a]pyrene, can be mineralized by nitrogen starved cultures of this fungus (Table Ia). Nearly coincidental degradation studies of a polychlorinated biphenyl (PCB) mixture (Aroclor 1254) by Eaton (5) using this microorganism produced similar results and conclusions. It is interesting to note that lignin, chlorinated lignin, and the overwhelming majority of lignin-derived low molecular weight by-products are already partially oxidized. In contrast, most of the compounds examined in our studies (3,4) and Eaton's (5) were highly reduced, having only an aromatic pi electron cloud, a hydrogen or a typically unreactive chlorine substituent available for attack. This demonstrates that P. chrysosporium is able to catalyze the initial oxidative step in the biodegradation of many highly reduced compounds. This step is important because the initial oxidation of an environmentally persistent xenobiotic is often (though not always) the most difficult step and a rate limiting step in its overall biodegradation. Thus, unlike many microorganisms which typically catalyze one or a few reactions during the microbial degradation of organopollutants, P. chrysosporium is able to initiate degradation of many organopollutants and to catalyze all of the steps in their complete degradation pathways to carbon dioxide.

Potential Advantages to the use of White Rot Fungi in Waste Treatment Systems. The potential usefulness of the white rot fungus P. chrysosporium in waste treatment systems has been discussed in greater detail elsewhere (3). Briefly the potential advantages to using this microorganism are: (1) The system normally attacks an insoluble substrate. (2) The lignin degrading system is relatively non-specific, thus it may be useful in the treatment of mixtures of organopollutants. (3) P. chrysosporium is a successful competitor, especially when wood based products are a major carbon source. (4) Biodegradation is not dependent on prior exposure to lignin or to a specific organopollutant. (5) Biodegradation of lignin proceeds to zero levels and the end product is carbon dioxide. This is precisely the type of degradation one would try to achieve for unwanted xenobiotics.

Perhaps the most interesting aspect concerning the use of this microorganism in the biological destruction of organopollutants, is the fact that it is able to dechlorinate chlorinated organic compounds. Unfortunately, dechlorination of organochlorides per se by P. chrysosporium has not been well studied. Our information concerning this process comes from the following: (1) In studies of the decolorization of the alkaline extract (E_1) obtained from a Kraft bleach plant, it was demonstrated that many of the organochlorides originally present were removed by treatment with P. chrysosporium in a rotating biological contactor (6,7). Furthermore, most

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch028

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch028

Table Ia. Mineralization of Selected ^{14}C-labeled Compounds by *Phanerochaete chrysosporium*.[a]

	Mineralization[b] (pmoles)	% Mineralization
Lindane	267.6	21.4
Benzo(a)pyrene	171.9	13.8
DDT	116.4	9.3
2,3,7,8,-TCDD	49.5	4.0
3,4,4',4'-TCB	25.1	2.0
2,4,5,2',4',5'-HCB[c]	86.0	1.7

Table Ib. Mineralization of ^{14}C-Labeled Biphenyl and Two Polychlorinated Biphenyl Mixtures.

	Mineralization (pmoles)	% Mineralization
Biphenyl	455.8	36.5
Aroclor 1242	256.3	20.5
Aroclor 1254	224.4	18.0

Table Ic. Mineralization of ^{14}C-Labeled Hexachlorobenzene and Pentachlorophenol.

	Mineralization (pmoles)	% Mineralization
Pentachlorophenol	570.4	45.6
Hexachlorobenzene	---	<1.0

Table Id. Mineralization of Chlordane and Mirex.

	Mineralization (pmoles)	% Mineralization
Chlordane[c]	607.7	12.2
Mirex	30.2	2.4

[a]Culture conditions were the same as those described in ref. 2, except that supplemental glucose (56 mM) was added to each culture after 30 days of incubation and incubations were continued for a total of 60 days. The $^{14}CO_2$ evolution assay was performed as described in ref. 2. DDT, 2,3,7,8-TCDD, 3,4,3',4'-TCB, 2,4,5,2',4'-5'-HCB, biphenyl, Aroclor 1242, Aroclor 1254, pentachlorophenol and hexachlorobenzene were uniformly labeled (^{14}C) in aromatic ring carbons atoms. Lindane, Chlordane and Mirex were uniformly labeled (^{14}C). Benzo[a]pyrene was labeled (^{14}C) in the 7 and 10 position. Except for hexachlorobenzene (purity = 96%), the radiochemical purity of the compounds used in this study was 97% or better.

[b]pmoles of ^{14}C-labeled substrate evolved as $^{14}CO_2$.

[c]The initial concentration of 2,4,5,2',4',5'-HCB and Chlordane was 5 nanomoles per culture. The initial concentration of all other compounds under study was 1.25 nanomoles/culture.

available evidence to support this position. Furthermore, reductive dechlorination of otherwise unsubstituted halogenated aromatic compounds under aerobic conditions would be predicted to be an unlikely event. An alternative method by which dechlorination could reasonably take place could entail direct substitution of chlorine by a hydroxyl group (31-33). This is an attractive hypothesis as the product can be considered to be a substituted phenol. This is important because chlorinated phenols are known to undergo dehalogenation in some systems (34,35). A similar pathway for dehalogenation might involve hydroxylation of one of the non-chlorinated carbon atoms (in a PCB, for example) followed, again, by dehalogenation of the phenol product. Still another mechanism of dechlorination requires that aromatic ring cleavage occur followed by dechlorination of the alkyl halide formed as a ring cleavage product. This appears to be the most common method of dehalogenation in nature (36-39). The mechanism(s) by which dechlorination is accomplished by P. chrysosporium and the role of the H_2O_2 dependent extracellular oxidases secreted by this fungus is at present unknown, but is the subject of current investigation.

In addition to studies demonstrating mineralization of the compounds presented in Table Ia, we have studied mineralization of other organic compounds in nutrient nitrogen deficient cultures of P. chrysosporium, in order to gain information concerning structural effects on biodegradation. From these studies the following generalization can be made: (1) Chlorination of an organic compound inhibits, but does not prevent, mineralization. This is seen most graphically when one compares the mineralization of biphenyl with the PCB mixtures Aroclor 1242 and Aroclor 1254 (Table Ib). Both Aroclor mixtures were mineralized but at a slower rate than biphenyl. However, comparison of our data regarding mineralization of the chlorinated compounds present following treatment were found in significantly reduced concentrations and some appeared to be metabolites of compounds present before treatment. In general, these metabolites appeared to contain fewer chlorine substituents than their presumptive precursors (6,7). (2) Studies in our laboratory (2,3) demonstrated that Lindane, an aliphatic compound that is chlorinated at every carbon atom, is degraded to CO_2 by this fungus, thus, demonstrating, albeit indirectly, that aliphatic dechlorination occurs. In another experiment we presented direct evidence for aliphatic dechlorination by showing that DDT undergoes reductive dechlorination to form DDD (2,4). (3) Eaton's work also provides evidence that supports the hypothesis that P. chrysosporium has the ability to dechlorinate organic compounds (5). In his study of the degradation and mineralization of ^{14}C-labeled Aroclor 1254, a PCB mixture, it was shown that after 19 days of incubation with nutrient nitrogen deficient cultures of P. chrysosporium, considerable non-polar radiolabeled material could be extracted which did not contain chlorine as measured by GLC using a Hall detector (5).

Because compounds like PCB are mineralized by P. chrysosporium, it is reasonable to suggest that these otherwise unsubstituted chlorinated aromatic compounds might undergo reductive dechlorination (substitution of Cl by H). There is, however, no currently

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch028

of individual PCB congeners (3,4,3',4'-TCB and 2,4,5,2',4',5'-HCB) with that of the Aroclor mixtures indicated that these individual congeners were mineralized much more slowly than the mixtures. The reason for this apparent anomalous finding is unknown. However, it may be due to the inherent stability of the two congeners selected for study. (2) The presence of at least one substituent other than chlorine appears to be required for the mineralization of chlorinated aromatic compounds as demonstrated by the comparison of hexachlorobenzene (HCB) mineralization and pentachlorophenol mineralization (Table Ic). HCB did not appear to be mineralized by P. chrysosporium whereas pentachlorophenol, a compound that differs from HCB only by the presence of a hydroxyl group in the place of one of the chlorine atoms, was extensively mineralized by this fungus. The fact that PCBs are mineralized demonstrates that hydrogen atom substituents are sufficient to allow the initial oxidation of the molecule. The recalcitrance of HCB is likely related to the stable nature of aromatic carbon-chlorine bonds and the probability that the highly electronegative chlorines coupled with their uniform symmetry in the HCB molecule prevents oxidation of the aromatic ring. (3) Chlorinated aliphatic compounds were also mineralized by P. chrysosporium. Interestingly, no generalizations can yet be made comparing the rate and extent of mineralization of chlorinated aliphatics with those of chlorinated aromatic compounds. For example, mineralization of Lindane (Table Ia) was slightly more rapid and extensive than Aroclor 1254 or Aroclor 1242 (Table Ib). However, mineralization of pentachlorophenol (Table Ic) was much more rapid and extensive than Lindane. In addition to Lindane (Table Ia), we have studied mineralization of two other polychlorinated aliphatic insecticides, Chlordane and Mirex (Table Id). Of all the non-aromatic organochlorines studied, Mirex was the most poorly mineralized. This lack of reactivity probably is due to the fact that this compound contains no hydrogen atoms and consists solely of C-C and C-Cl bonds. (4) In addition to hexachlorobenzene, the heterocyclic herbicide Atrazine was the only other compound in our study which was refractory to ring cleavage as assayed by mineralization of ^{14}C-ring labeled carbon atoms (data not presented). Apparently, ring nitrogen atoms in this heterocyclic compound prevent attack by the aromatic ring cleavage system(s) of this fungus.

Conclusions

The white rot fungus, Phanerochaete chrysosporium has the ability to degrade and mineralize many structurally diverse chlorinated organic compounds (2-7). Studies in our laboratory and by others demonstrate that degradation and mineralization is dependent, at least in part, on the lignin degrading system of this fungus that is expressed in response to nutrient starvation (2-7).

The ability to degrade a wide variety of chlorinated organic compounds coupled with the fact that the initial oxidation of these compounds is likely to be extracellular suggests that this system may be of value in the microbial treatment of materials contaminated with these compounds.

American Chemical Society
Library
1155 16th St., N.W.
Washington, D.C. 20036

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch028

Acknowledgments

This work was supported by Cooperative Agreement #CR811464, U.S. Environmental Protection Agency, Office of Research and Development, Hazardous Waste Engineering Research Laboratory, Cincinnati, OH, P.R. Sferra, Project Officer. The authors thank Teresa Vollmer for secretarial assistance in the preparation of this manuscript.

Literature Cited

1. Murphy, S. D. In "Casarett and Doull's Toxicology: The Basic Science of Poisons"; Doull, J.; Klaassen, C. D.; Amdur, M. O., Eds.; MacMillan: New York, 1980; pp. 357-408.
2. Bumpus, J. A.; Tien, M.; Wright, D; Aust, S. D. Science 1985, 228, 1434-6.
3. Bumpus, J. A.; Tien, M.; Wright, D. S.; Aust, S. D. Proc. 11th Ann. Res. Symp., 1985, USEPA, EPA/600/9-85/028, pp. 120-6.
4. Bumpus, J. A.; Aust, S. D. Proc. International Conference New Frontiers for Hazardous Waste Management, 1985, USEPA, EPA/600/9-85/025, pp. 404-10.
5. Eaton, D. C. Enzyme Microb. Technol. 1985, 7, 194-6.
6. Chang, H.-M.; Joyce, T. W.; Campbell, A. G.; Gerrard, E. D.; Huynh, V. B., Kirk, T. K. In "Recent Advances in Lignin Biodegradation"; Uni Publishers: Tokyo, 1983; pp. 257-68.
7. Huynh, V. B.; Chang, H.-M.; Joyce, T. W.; Kirk, T. K. T.A.P.P.I. J. 1985, 68, 98-102, 7.
8. Crawford, R. In "Lignin Biodegradation and Transformation"; John Wiley and Sons: New York, 1981; p. 154.
9. Forney, L. J.; Reddy, C. A.; Tien, M.; Aust, S. D. J. Biol. Chem. 1982, 257, 11455-62.
10. Hall, P. Enzyme Microb. Technol. 1980, 2, 170-6.
11. Amer, G. I.; Drew, S. W. Dev. Ind. Microbiol. 1980, 22, 479-84.
12. Nakatsubo, F.; Reid, I. D.; Kirk, T. K. Biochem. Biophys. Res. Commun. 1981, 102, 484-91.
13. Gold, M. H.; Kutsuki, H.; Morgan, M. A. Photochem. Photobiol. 1983, 38, 647-51.
14. Kutsuki, H.; Enoki, A.; Gold, M. H. Photochem. Photobiol. 1983, 37, 1-7.
15. Kutsuki, H.; Gold, M. H. Biochem. Biophys. Res. Commun. 1982, 109, 320-7.
16. Koenigs, J. W. Arch. Microbiol. 1974, 99, 129-45.
17. Koenigs, J. W. Phytopath. 1972, 62, 100-10.
18. Tien, M.; Kirk, T. K. Science 1983, 221, 661-3.
19. Tien, M.; Kirk, T. K. Proc. Natl. Acad. Sci. USA 1984, 81, 2280-4.
20. Andersson, L. A.; Renganathan, V.; Chiu, A. A.; Loehr, T. M.; Gold, M. H. J. Biol. Chem. 1985, 260, 6080-7.
21. Gold, M. H.; Kuwahara, M.; Chiu, A. A.; Glenn, J. K. Arch. Biochem. Biophys. 1984, 234, 353-62.
22. Huynh, V. B.; Crawford, R. L. FEMS Microbiol. Lett. 1985, 28, 119-23.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch028

23. Kirk, T. K.; Mozuch, D.; Tien, M. Biochem. J. 1985, 226, 455-60.
24. Paszczynski, A.; Huynh, V. B.; Crawford, R. L. Arch. Biochem. Biophys. 1986, 244, 750-65.
25. Harvey, P.J.; Schoemaker, H. E.; Bowen, R. M.; Palmer, J. M. FEBS Lett. 1985, 183, 13-16.
26. Schoemaker, H. E.; Harvey, P. J.; Bowen, R. M.; Palmer, J. M. FEBS Lett. 1985, 183, 7-12.
27. Kuwahara, M.; Glenn, J. K.; Morgan, M. A.; Gold, M. H. FEBS Lett. 1984, 169, 247-50.
28. Glenn, J. K.; Gold, M. H. Arch. Biochem. Biophys. 1985, 242, 329-41.
29. Paszczynski, A.; Huynh, V. B.; Crawford, R. L. FEMS Microbiol. Lett. 1985, 29, 37-41.
30. Lundquist, K.; Kirk, T. K.; Connors, W. J. Arch. Microbiol. 1977, 112, 291-6.
31. Johnston, H. W.; Briggs, G. G.; Alexander, M. Soil Biol. Biochem. 1972, 4, 187-90.
32. Klages, U.; Ligens, F. FEMS Microbiol. Lett. 1979, 6, 201-3.
33. Marinucci, A. C.; Bartha, R. Appl. Environ. Microbiol. 1979, 38, 811-17.
34. Chu, J.; Kirsch, E. J. Appl. Microbiol. 1972, 23, 1033-35.
35. Chu, J.; Kirsch, E. J. Dev. Ind. Microbiol. 1973, 14, 264-73.
36. Goldman, P.; Milne, G. W. A.; Pignataro, M. T. Arch. Biochem. Biophys. 1967, 118, 178-84.
37. Hartman, J.; Reineke, W.; Knackmuss, H.-J. Appl. Environ. Microbiol. 1979, 37, 421-8.
38. Reineke, W.; Knackmuss, H.-J. J. Bacteriol. 1980, 142, 467-73.
39. Reineke, W. In "Microbial Degradation of Organic Compounds"; Marcel Dekker, Inc.: New York, 1984; pp. 319-60.

RECEIVED November 25, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch028

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch029

Chapter 29

Photochemical Surface Decontamination: Application to a Polychlorinated Biphenyl Spill Site

William M. Draper[1,4], Robert D. Stephens[2], and Luis O. Ruzo[3]

[1]Environmental Health Division, School of Public Health, University of Minnesota, Minneapolis, MN 55455
[2]Hazardous Materials Laboratory, California Department of Health Services, 2151 Berkeley Way, Berkeley, CA 94704
[3]Pesticide Chemistry and Toxicology Laboratory, Department of Entomological Sciences, University of California—Berkeley, Berkeley, CA 94720

A photochemical treatment process is evaluated for decontamination of PCB-tainted surfaces. In the laboratory commercial PCB mixtures were photochemically reactive under fluorescent "sunlight" (maximal output 300 nm) but not "blacklight" lamps (maximal output 360 nm). PCB were photolabile in either organic solvents or neat thin films indicating that supplemental H-donors were not required for reactivity. A prototype surface photoreactor constructed of a Flexiform aluminum frame, lamp fixtures and Westinghouse FS40 fluorescent lamps irradiated a 1.5 m^2 surface with a light intensity of 4,600 uW/cm^2. In a field test on concrete contaminated with Aroclor 1260 about 10 years ago, the prototype reactor destroyed 47% of the PCB residue after 21 hr of treatment. Means for improving the destruction efficiency are discussed.

Polychlorinated biphenyls (PCB) are noted for their stability. They are resistant to heat, oxidation and attack by strong acids and bases. As a result of these extraordinary properties PCB have been used widely and successfully as dielectric fluids, fire retardants, heat transfer agents, hydraulic fluids, plasticizers and in other applications (1).

[4]Current address: California Public Health Foundation, 2151 Berkeley Way, Berkeley, CA 94704

0097-6156/87/0338-0350$06.00/0
© 1987 American Chemical Society

We now know that PCB are hepatotoxic and that they impair reproductive function and produce cancer in experimental animals (2,3). PCB undergo food chain "biomagnification" and accumulate in human and animal tissues, sometimes exceeding acceptable levels. As a result of the once valued stability and potential for chronic or delayed toxicity, widespread environmental contamination by PCB is now viewed as a threat to both environmental quality and public health (4).

The 1977 U. S. Environmental Protection Agency (EPA) ban on PCB manufacture greatly curtailed occupational PCB exposure. Potentially hazardous exposures continue, however, for workers in spill clean-up, servicing and dismantling of transformers and capacitors, electrical utility work, and firefighting. A tremendous volume of PCB is still in use and numerous sites in this country and abroad remain contaminated with PCB oil.

In contrast to their chemical stability, PCB are remarkably reactive when exposed to ultraviolet (UV) light. Over the past several decades the photoreductive dehalogenation reactions of PCB have been examined in organic solvents (5,6), neat thin films and solid phase (7,8) and as surface deposits on silica (9).

The "classical" photoreduction mechanism involves the formation of free radicals by homolytic cleavage. Absorbed light energy (hv) is transformed to electronic energy in a π to π^* transition. In the electronically-excited state (indicated by an asterisk) the carbon-chlorine bonds undergo fission yielding aromatic radicals and chlorine atoms, both of which abstract hydrogen atoms from the solvent yielding intermediate photoproducts (Figure 1). The photochemical sequence is repeated ultimately yielding the chlorine free biphenyl nucleus. Alternate photoreductive dechlorination mechanisms involving heterolytic cleavage of C-Cl bonds (10) and electron transfer (11) have been proposed.

The principal direction of the published photochemical research to date has been to elucidate pathways which might be responsible for the degradation of these refractory substances in nature. This basic research has not been extensively applied either to on- or off site treatment of hazardous substances. In this chapter we extend the existing PCB photochemistry literature by considering this potential. The development and fabrication of a prototype surface photoreactor, and its preliminary evaluation at a PCB-contaminated site are described. Photochemical treatments may be applicable to many other classes of hazardous chemicals as well including the polychlorinated dibenzodioxins (PCDD) and dibenzofurans (PCDF) about which this monograph is chiefly concerned.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch029

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch029

Methods and Materials

Chemicals. Analytical reference standards for Aroclor 1254 and 1260 were provided by the U. S. EPA (Research Triangle Park, NC). Solvents were commercially available, pesticide grade and were used as received.

Irradiation. Solutions were irradiated in sealed borosilicate or quartz test tubes held in a merry-go-round apparatus ensuring uniform illumination. A Rayonet Model RPR-100 photoreactor (Southern New England Ultraviolet Co., Hamden, CT) equipped with 16 RPR-3500 "blacklight" lamps or 16 RPR-3000 "sunlight" lamps was used in preliminary laboratory studies of PCB photolability. For solution phase experiments a 1.0 mg/L petroleum ether solution of Aroclor 1254 was studied. Periodically, irradiated samples were subjected to direct analysis by capillary electron capture gas liquid chromatography (EC-GLC). The photolability of PCB on surfaces was determined by irradiation of neat Aroclor 1254 thin films (18 ug/cm^2) on glass Petri plates fitted with borosilicate glass covers. After irradiation plates were withdrawn from the photoreactor and the PCB residue dissolved in 2 X 5 mL of hexane for EC-GLC analysis.

Actinometry. The trifluralin actinometer was used to measure light intensity from polychromatic sources (12,13). Table I. provides Westinghouse FS40 spectral energy distribution and irradiance data and trifluralin extinction coefficients needed for actinometry. For the sunlight phosphor lamps the actinometry procedure was modified by using isooctane as the solvent and quartz cells, both transparent to wavelengths emitted by these lamps. Calculations were adjusted for the higher reaction quantum yield of trifluralin in isooctane (Φ = 0.018) relative to toluene (Φ = 0.0091) (12).

Sampling PCB on Concrete. A template was used to demarcate 10 X 10 cm squares on contaminated concrete surfaces. Isooctane (1.5 mL) was applied and after about 45 seconds the solvent was imbibed on two filter paper disks. The filter paper disks were held in stoppered, prerinsed test tubes for shipment to the laboratory. Filter paper disks were extracted by immersion in 40 mL of petroleum ether for one hr and the extracts dried (NaCl) and diluted with isooctane prior to analysis.

Chemical Analysis. All chemical measurements were made by EC-GLC using a 15 m X 0.53 mm (ID) fused silica capillary (DB-5, J & W Scientific) mounted in a packed injection port with the following conditions: inlet temperature, 250^{o}C; detector temperature, 300^{o}C; He

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch029

Table I. Emission Characteristics and Spectral Irradiance Values for Westinghouse FS40 Lamp and Trifluralin Absorption Coefficients[a]

Wavelength (nm)	Spectral Energy Distribution, %	Spectral Irradiance Joules/s/cm^2	Spectral Irradiance Photons/s/cm^2	Trifluralin Extinction Coefficients, ε
265	0.24	1.1E-5	1.5E13	6,420
275	1.3	5.8E-5	8.0E13	7,425
285	5.0	2.3E-4	3.3E14	6,260
295	16	7.2E-4	1.1E15	3,750
305	21	1.0E-3	1.5E15	2,210
315	19	8.9E-4	1.4E15	1,500
325	14	6.3E-4	1.0E15	1,460
335	7.8	3.6E-4	6.1E14	1,500
345	4.2	1.9E-4	3.3E14	1,670
355	1.8	8.3E-5	1.5E14	1,840
365	2.1	9.2E-5	1.7E14	2,060
375	0.64	2.9E-5	5.5E13	2,230
385	0.40	1.8E-5	3.5E13	2,320
395	0.32	1.5E-5	3.0E13	2,320
405	1.9	8.7E-5	1.8E14	2,320
415	0.32	1.5E-5	3.1E13	2,260
425	0.32	1.5E-5	3.2E13	2,160
435	3.7	1.7E-4	3.7E14	1,960
445	0.16	7.3E-6	1.6E13	1,660
455	0.16	7.3E-6	1.7E13	1,290

[a] Spectral irradiance values are based on a total radiant intensity (250-460 nm) of 4,600 uW/cm^2, a typical light flux for the operating reactor as determined by the trifluralin actinometer. The FS 40 lamp has negligable output at 253.7 nm and several minor emission bands above 500 nm in a region trifluralin does not absorb.

carrier gas flow rate, 7 mL/min; detector purge gas flow rate, 60 mL/min Ar-methane (95:5, v/v). Trifluralin eluted in 2.7 min with an oven temperature of 195°C. At 220°C the following peaks were used to quantitate Aroclor 1260: 1.21; 1.40; 1.65; 1.84; 2.52; and 3.03 (retention times relative to p,p'-DDE which eluted in 8.2 min). Determinations of Aroclor 1254 were based on the following peaks: 0.75; 0.85; 1.05; 1.22; 1.43; and 1.65. Quantitation was achieved by suming peak areas with absolute calibration relative to external Aroclor standards.

Fabrication of Surface Photoreactor. A prototype surface photoreactor was constructed using commercially-available, 1.2-meter fluorescent lamp fixtures mounted on an aluminum Flexiform frame. The configuration consisted of two fixtures holding four 40-watt, Westinghouse FS40 lamps mounted in parallel. The enamel surfaces of the canopy were covered with aluminum foil to increase the reflectance of UV and near UV radiation. With the lamps suspended 10 cm from the contaminated concrete a 1.5 m^2 surface was exposed to intense light.

Field Test. The University of Minnesota Rosemount Research Center (RRC) is listed on the U. S. EPA Superfund National Priorities List for hazardous waste disposal site cleanup and is one of two university-owned sites so designated (14). The RRC, briefly operated as the federal government's Gopher Ordinance Plant in the second world war, was converted by the university to an agricultural research facility. Areas were designated for disposal of university-generated solvents and chemicals, and some facilities were leased to private companies. Several areas at the RRC are contaminated with PCB; in particular, Aroclor 1260 has been detected in samples taken from the site. Between August, 1973 and January, 1978 buildings and grounds now contaminated with PCB were leased to a private firm engaged in servicing and dismantling transformers and other electrical equipment. Other commercial concerns dealing in used equipment, also spread PCB oil at RRC, and some waste transformer oils were incinerated.

The extent and distribution of PCB contamination at RRC was investigated by the Minnesota Pollution Control Agency in 1978, and again in 1984 by a contract laboratory as part of a site remedial investigation (15). The facility features highly contaminated gravel-covered areas and debris pits. Soil profiles in discolored areas indicated Aroclor 1260 levels of 0.14-0.41% (w/w). A concrete building floor also was contaminated with Aroclor 1260, but at comparatively low levels. The measured PCB contamination of these concrete surfaces exceeded levels deemed safe by the Minnesota Department of Health. The applicable state

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch029

standard at work sites is 50 ug PCB/100 cm2 as determined by a wipe test of the type used here.

The field test consisted of decontaminating the PCB-tainted concrete surface. Before and after 21 hr of photochemical treatment a 1.2 m^2 area was sampled extensively (n = 8) to quantify PCB residues. Treated and control samples were located on alternate positions of a grid to minimize sampling error.

Results

Energy Requirements for PCB Photodecomposition. PCB photodegrade very slowly on irradiation with fluorescent blacklight lamps. After 48 hr of irradiation (8,600 uW/cm^2) 87% of the Aroclor 1254 was recovered from petroleum ether solutions. In contrast, PCB are highly reactive on exposure to fluorescent sunlamps (Figure 2). Aroclor 1254 irradiated in Pyrex tubes that are opaque to the RPR-3000's 253.7 nm emission line had a half-life of one hr. The photolysate mixtures exhibit a pungent, noxious odor indicating the formation of volatile photoproducts, possibly chlorine or low molecular weight chlorinated hydrocarbons derived from free-radical chlorination of the solvent. Similar photochemical chlorination of the solvent was observed by Crosby and Hamadmad (16) on irradiation of pentachloronitrobenzene in hexane.

Differences in PCB photolability are attributed to the emission spectra of the lamps tested (Figure 3). The blacklight lamp has a UV cutoff of about 310 nm whereas the sunlamp has considerable output in the 270-300 nm range and as much as 15% of the radiant energy at the 253.7 nm mercury line. Because the borosilicate cells are opaque below about 285 nm we conclude that the observed photoreactivity is due to near UV light in the 285-300 nm wavelength range. When quartz cells were used in the laboratory photoreactor the Aroclor 1254 half-life dropped to 12 min. In general then, the efficacy of light for PCB treatment is ranked as follows: UV (253.7 nm) > near UV (285-300 nm) >>> near UV (>300 nm). Light absorption alone may account for these differences as the 253.7 nm Hg line corresponds roughly to PCB absorption maxima.

The presence of a hydrogen-donating solvent is not essential for PCB photoreactivity. Neat Aroclor 1254 thin films on glass (18 $ug/100\ cm^2$) were photolabile with surface deposits exhibiting half-lives of ~50 min.

Prototype Photoreactor. The Westinghouse FS40 sunlight lamp envelope does not transmit the 253.7 nm Hg line and has its major emission band shifted about 10 nm toward higher wavelengths relative to the RPR-3000 lamp (Figure 3). Light output from the prototype reactor was increased 38% on covering the white enamel fixtures with metal foil. The light intensity under the

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch029

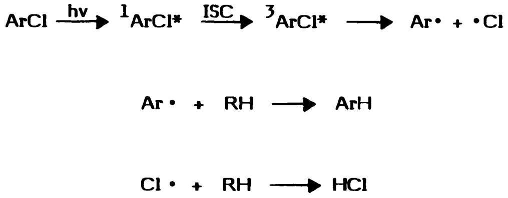


Figure 1. Generalized photoreduction scheme for haloarenes.

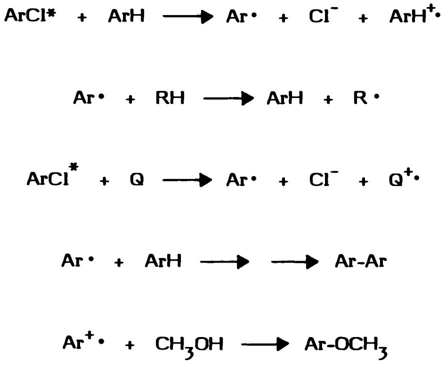


Figure 2. Photodecomposition of Aroclor 1254 in petroleum ether solutions irradiated with fluorescent sunlight lamps (borosilicate cells).

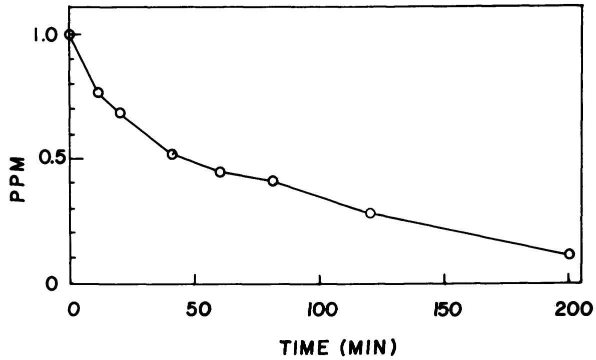


Figure 3. Normalized emission spectra for sunlight and blacklight, mercury-excited phosphor lamps.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch029

In Solving Hazardous Waste Problems; Exner, J.;
ACS Symposium Series; American Chemical Society: Washington, DC, 1987.

photoreactor canopy at the irradiated surface was 4,600 uW/cm^2, well below values measured in the laboratory photoreactor which were typically 11,800 and 24,500 uW/cm^2 with blacklight and sunlight lamps, respectively. The fluorescent lamps studied emit very little heat and under operating conditions the irradiated surface was not appreciably elevated in temperature. The shift in emission spectrum in combination with reduced light output indicated that reactions would proceed much more slowly under the surface reactor when compared to the laboratory system.

Field Test. Before treatment the concrete floor was contaminated with 81 ± 31 ug Aroclor 1260/100 cm^2 (n = 8). The high relative standard deviation has contributions from surface PCB distribution (sampling error), PCB recovery during sampling and precision in quantitation. Because the analytical method had few steps, sample concentrations were similar, and the EC detector was operated in a linear region, analytical error was probably a minor contributing factor. A fine layer of dust and soil was present on the concrete, but no surface preparation was undertaken prior to the test.

After 21 hours of irradiation the concrete was again sampled in close proximity to pretreatment samples (Figure 4). Post-treatment Aroclor 1260 levels were 43 ± 13 ug/100 cm^2 (n = 8). The 47% reduction in PCB concentration is statistically significant at the 99% confidence level.

The field test of this photochemical reactor to our knowledge represents the first demonstration of a photochemical treatment process for in-situ remedial cleanup of PCB contaminated surfaces. At this site PCB oils were deposited 7 to 13 years prior to treatment and represent residues weathered by volatilization and allowed to thoroughly migrate in the concrete surface layers. It is expected that overlying dust and soil may have provided shade, possibly reducing the PCB destruction efficiency. PCB sorbed on particulate matter would react more slowly due to reduced light penetration.

Discussion

The Photochemistry of PCB. The light-mediated reactions of PCB and their analogs have received considerable attention and several reviews of PCB photochemistry are available (1,17,18). Rappe (19) has reviewed the photochemistry of halogenated aromatics in general.

PCB absorb UV light efficiently, but absorption of near UV light is relatively weak. The long wavelength UV absorption band of biphenyl and its meta- and para-substituted derivatives appears at ~250 nm (ε =

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch029

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch029

20,000). The absorption of light by PCB is determined by their geometry (20,21). Ortho-chlorines lower ε drastically while para-chlorines have the opposite effect. When the biphenyl conformation is twisted by substituents there is reduced overlap in molecular orbitals increasing the energy of the excited state and shifting absorption maxima to lower wavelengths. UV absorbance also increases with the degree of chlorination. Decachlorobiphenyl, for example, absorbs strongly at ~300 nm (22).

Photochemical reactivity is affected both by the rate of light absorption and reaction quantum yield (Φ), that fraction of molecules reacting per photon absorbed. Quantum yields for individual PCB isomers vary tremendously (21,23) although, in general, they are quite high in comparison to other pollutants. PCB with para- and particularly ortho- chlorines react most rapidly (see 2,2',4,4'-tetrachlorobiphenyl, Table II.). As a general rule halogenated aromatic molecules including PCB react far more rapidly in hydrocarbon solvents than in water, i.e., Φ for TCDD is 20 times higher in hexane than water (24). There are exceptions to this generality, however. 3,4-Dichloroaniline and monochloro aromatics react as fast or faster in water because cationic and not radical intermediates are involved (24, 25). In cyclohexane Φ range from 0.002 to 0.1 and in aqueous acetonitrile PCB quantum yields as high as 0.25 have been reported (Table II.). Reaction rates in methanol are greater than in cyclohexane (21).

The majority of PCB photodegradation studies have been carried out in solution with organic or combined solvents preferred due to their low water solubility. Reductive dechlorination is the predominant photodegradation pathway in hydrocarbon solvents as well as alcohols. In aqueous media and alcohols photonucleophilic displacement reactions yielding phenols or alkoxy derivatives, respectively, also are detected. In the gas phase PCB are photochemically converted to hydroxylated derivatives (26). Photochemical reduction also is the major reaction pathway in thin films (7), solid phase (8) and on silica gel surfaces (9).

Not all chlorines in PCB structures are equally reactive. Ortho-chlorines are removed preferentially to those in meta- and para-positions (21,23). Thus, 2,2',4,4'-tetrachlorobiphenyl yields sequentially 2,4,4'-trichloro- and then 4,4'-dichlorobiphenyl. Deuterium also is incorporated preferentially in the ortho-positions when deuterated solvents are employed (27) demonstrating H abstraction from the solvent.

As noted earlier there are various photoreductive dechlorination mechanisms. The triplet excited state is believed to undergo homolytic cleavage in the case of ortho-chlorinated PCB, a hypothesis supported by

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch029

Table II. Extinction Coefficients and Reaction Quantum Yields for PCB[a]

PCB Isomer/Mixture	Extinction[b] Coefficient	Quantum[c] Yield
2,2',4,4'-Cl_4	140	0.10
2,2',3,3'-Cl_4	116	0.007
2,2',5,5'-Cl_4	120	0.010
2,2',6,6'-Cl_4	30	0.006
3,3',5,5'-Cl_4	1410	0.002
3,3',4,4'-Cl_4	6740	0.005
4-Cl	120	0.004
2,4-Cl_2	25	0.22
2,4,6-Cl_3	7	0.25
2,2',5,5'-Cl_4	30	0.017
Cl_{10}	1200	>0.21
Aroclor 1232	60	0.036
Aroclor 1268	770	0.16

a According to references 21 and 22.

b Extinction coefficients for the tetrachloro series were measured at 290 nm. Extinction coefficients for the remaining isomers and mixtures were determined at 300 nm.

c Quantum yields for the tetrachloro series were determined in cyclohexane irradiated at 300 nm. The second series of isomers and mixtures were irradiated in acetonitrile-water (4:1, v/v) with 254 nm light.

heavy atom effects (28,29) and phosphorescence studies (23). PCB generally exhibit low fluorescence quantum yields (22), but phosphoresce efficiently (21) due to high intersystem crossing yields from the singlet to the triplet excited states. Dechlorination of the *meta*- and *para*- isomers may involve excimer formation (29). In condensed PCB films the major photoprocesses probably include e^- transfer reactions leading to both dechlorination and polymerization.

Photolysis of Related Haloaromatics. Many other classes of hazardous materials may be susceptible to *in situ* photochemical treatment. Polybromobiphenyls (PBB) photodecompose more readily than PCB due to the weaker C-Br bonds. The major reaction is reductive debromination but quaterphenyls also are formed in low yield (30). *Ortho*-bromines are cleaved preferentially for pure isomers but apparently not in commercial PBB mixtures (29).

Chlorinated terphenyls are reductively dechlorinated, but also undergo substantial photosubstitution in alcohol solvents (31) in contrast to the low yield of substitution products from PCB. In the terphenyl series photosubstitution is believed to take place by attack of the nucleophile directly on the excited state with concomitant chloride ejection. Alkylated PCB molecules (chloroalkylenes), candidate PCB replacement compounds, exhibit UV electronic spectra nearly identical to those of PCB yet photodegrade more rapidly (28). Another possible PCB substitute consists of a mixture of bis-(chlorophenyl)ethanes (Iralec) that also undergoes dechlorination and substitution reactions on irradiation (32).

Support for electron transfer processes leading to dehalogenation is provided in studies of chloronaphthalenes (33). Reductive dechlorination, binaphthyl formation and photonucleophilic reactions can be rationalized with the scheme depicted in Figure 5. Analogous electron transfer processes have been proposed for the cyclization/dehalogenation of polyhalogenated-2-phenoxyphenols (so-called "pre-dioxins") yielding PCDF and PCDD (34). The photocyclization of predioxins increases with solvent dielectric constant and, based on the dependence of Φ on concentration, intramolecular rather than intermolecular electron transfer appears to predominate (35).

Accelerated PCB Photodegradation. Sensitized or accelerated PCB photodegradation has been observed in many published reports suggesting possible applications in treatment of hazardous substances. Substantial rate enhancements have been obtained in photochemical reaction mixtures containing added amines as electron

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch029

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch029

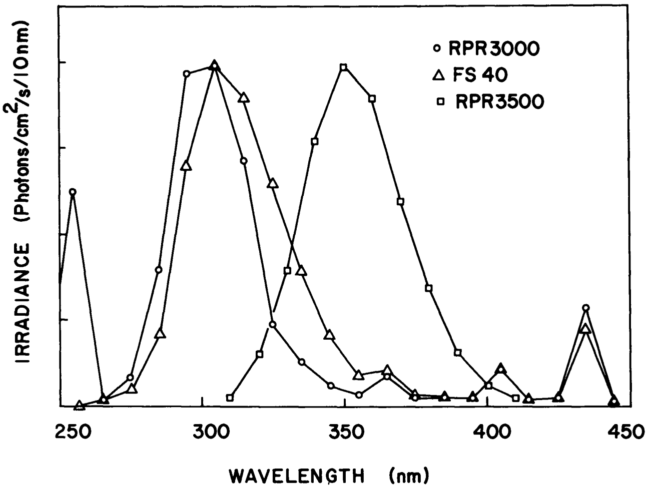


Figure 4. Photochemical decomposition of Aroclor 1260 on concrete. The rectangle denotes the photoreactor canopy. Pretreatment levels (ug/cm^2) are indicated with filled symbols, open symbols indicate PCB levels measured after 21 hr of light treatment.

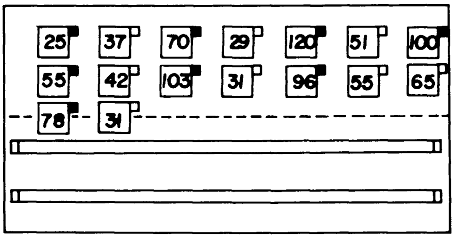


Figure 5. Electron transfer induced photochemical dehalogenation, dimerization and displacement. "Q" indicates a triplet quencher like cyclohexadiene.

transfer agents. The radical anion formed on initial electron transfer ($ArCl^{\cdot -}$) is believed to expel chloride directly, or undergo protonation prior to release of chlorine atoms (17). Triethylamine increases photocyclization of chlorinated phenoxyphenols (34,36), debromination of PBB (30), photoreduction of chloronaphthalenes (37), and dechlorination of PCB (22) and polychlorinated terphenyls (31). Triethylamine also sensitizes the photodechlorination of PCB on silica gel and clay minerals (38). Generally, these reactions are less effective for *ortho* chloro isomers and when these additives are used the isomeric photoproduct distribution changes (11). Other electron-rich molecules including aromatic amines, indoles and phenols are effective sensitizers for photodechlorination of chloroacetic acid (39).

Enhancement of PCB photodegradation also is observed in the presence of trifluoroacetic acid (40), presumably via the protonated intermediates ($ArClH^{+\cdot}$). Dienes, such as 1,3-cyclohexadiene, accelerate haloaromatic photoreactions (41) and modify chloronaphthalene photochemistry by enhancing reductive dechlorination and suppressing dimerization (42). The mechanism is believed to involve exciplex formation with the diene or protonation by the olefin (43).

Other compounds enhance PCB photodegradation including sodium borohydride (44), alkaline alcoholic solutions (45) and aqueous suspensions of titanium dioxide (46), a system also effective in treating pentachlorophenol (47).

Does Photochemical Treatment of PCB Reduce Health Hazard? As with any new technological process, and particularly in the decontamination of hazardous materials, potential health consequences need to be considered. Photochemical or other treatment processes for PCB may reduce or increase potential hazard because the composition and isomer distribution is altered. In the case of PCB three situations require special attention: 1) the potential for photochemical formation of PCDF and PCDD or other extremely toxic products; 2) the selective photochemical destruction of these substances in PCB mixtures; and 3) shifts in PCB congener distribution to structures with exceptional toxicity.

There are several examples of the first situation in the literature. Predioxin contaminants in PCB mixtures (48) undergo photochemical ring closure to PCDD on irradiation (34,49). Chlorinated diphenyl ethers, also trace PCB contaminants, are cyclized to PCDF on irradiation in hexane solution although photodechlorination predominates (50). Acetone sensitizes this photocyclization as well as that of polychlorophenols (35) suggesting that triplet excited

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch029

states are involved. Under alkaline conditions pentachlorophenol and other acidic ortho-chlorophenols have been reported to condense by way of photonucleophilic displacement reactions to PCDD (51).

The degree to which PCDF and PCDD accumulate in irradiated PCB mixtures will depend on many factors including the concentration of precursor molecules and, in turn, the photolability of the dibenzo products. In any event, the photochemical removal of PCDD and PCDF is likely to be far more efficient than PCB photodegradation itself. PCDD and PCDF are more photolabile than PCB because of their large absorption cross sections and high reaction quantum efficiencies. Chlorinated dibenzodioxins are extremely unstable in UV light in methanol, diesel oil, liquid phenoxy ester formulations and other organic media (52,53). Fortunately, chlorines in the lateral positions (i.e., 2-, 3-, 7-, 8-) of PCDD are preferentially cleaved on photoreduction (54) leading to the least toxic products. PCDF, like PCDD, appear to be quite photoreactive (55) and in these structures the chlorines para- to biphenyl linkage are most reactive (56). The high photochemical reactivity of PCDD and PCDF relative to PCB supports the view that, if formed, these toxic molecules will occur only in very low steady-state levels. This consideration, however, warrants further study.

The toxicity of PCB mixtures *in vivo* and *in vitro* is largely attributable to tetra-, penta- and hexachloro congeners that are capable of assuming a planar conformation (57,58,59). Mono ortho-chloro, coplanar congeners mimic the toxicological effects of 2,3,7,8-TCDD (57). Any photochemical or chemical treatment process which would substantially enrich a PCB deposit in these structures, regardless of its destruction efficiency, would be undesirable. PBB photoproduct mixtures are of increased toxicity due to this sort of isomer redistribution (60). In the case of PCB the preferential photochemical reaction of ortho-halogens may lead to intermediate mixtures of enhanced toxicity and this aspect of PCB reactivity warrants further study. As noted above the product distribution ratio changes markedly on going from direct to sensitized photolysis (11) and thus it may be possible to control the nature of the photolysate residue.

Conclusion

Photochemical treatments hold promise for on-site decontamination of hazardous substances on various surfaces including building walls and floors, machinery and electrical equipment (e.g., transformer cases). Further applications include cleaning equipment used in remedial activities at contaminated sites and in

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch029

routine small scale decontamination in chemical laboratories. The destruction efficiency on concrete may be improved by: 1) increasing the interception of light (i.e., by use of a solvent to desorb the pollutant, or prior removal of surface dust); 2) use of a photoreactor with increased light output, particularly UV output; and 3) possible use of hydrogen- or electron donating solvents or photochemical sensitizers. The present study demonstrates qualitatively that photochemical treatments are effective under field conditions. Further basic studies and engineering development will be needed to define the the ultimate destruction efficiency achievable, the applicability to other contaminants and surfaces, and the nature of the photolysate residue and its modification.

Acknowledgment

John Lulewicz assisted in the analysis of PCB and Danny Mar assisted in manuscript preparation. Fay Thompson (University of Minnesota) provided valuable discussions and arranged the field experiment at RRC. The authors gratefully acknowledge the support of the National Institute of Environmental Health Sciences (Grant Nos. R23 ES03524, WMD; P01 ES00049, LOR) and the National Institute for Occupational Safety and Health of the Centers for Disease Control (Grant No. R01 OH02119, WMD).

Literature Cited

1. Hutzinger, O.; Safe, S.; Zitko, V. The Chemistry of PCB's, CRC Press: Cleveland, 1974.
2. DHEW: Subcommittee on health effects of PCB's and PBB's.Environ. Health Perspect. 1978, 24, 146.
3. IARC: Working group on the evaluation of the carcinogenic risk of chemicals to humans - Polychlorinated biphenyls. 1978, 18.
4. "The Toxicology of PCBs: An Overview with Emphasis on Human Health Effects and Occupational Exposures"; State of California Department of Health Services/Department of Industrial Relations: Berkeley, CA, 1981.
5. Safe, S.; Hutzinger, O. Nature (London) 1971, 232, 641.
6. Ruzo, L.O.; Zabik, M.J.; Schuetz, R. D. Bull. Environ. Contam. Toxicol. 1972, 8, 217.
7. Hustert, K.; Korte, F. Chemosphere 1972, 1, 7.
8. Hutzinger, O.; Safe, S.; Zitko, V. Environ. Health Perspect. 1972, 1, 15.
9. Gab, S.; Nitz, S.; Parlar, H.; Korte, F. Chemosphere, 1975, 4, 251.
10. Mamantov, A. Chemosphere 1985, 14, 901.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch029

11. Soumillion, J. P.; DeWolf, B. J. Chem. Soc. Chem. Comm. 1981, 436.
12. Draper, W. M. Chemosphere 1985, 14, 1195.
13. Draper, W. M. In Photochemistry of Environmental Aquatic Systems; W. Cooper and R. Zika, Eds.; ACS Symposium Series 327; American Chemical Society: Washington, DC, 1986; 268-280.
14. Sanders, H. J. Chem. Eng. News 1986, 64, 21.
15. "Phase I Report - University of Minnesota Rosemount Research Center Remedial Investigation"; Soil Exploration, February, 1985.
16. Crosby, D. G.; Hamadmad, N. J. Agric. Food Chem. 1971, 19, 1171.
17. Bunce, N. J. Chemosphere 1982, 11, 701.
18. Zabik, M. J.; Leavitt, R. A.; Su, G. C. C. Ann. Rev. Entomol. 1976, 21, 61.
19. Rappe, C. In Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzofurans and Related Products; R. D. Kimbrough, Ed; Elsevier: New York, 1980.
20. Wagner, P.J. J. Amer. Chem. Soc. 1967, 89, 2820.
21. Ruzo, L.O., Zabik, M. J.; Schuetz, R. D. J. Agric. Food Chem. 1974, 22, 199.
22. Bunce, N. J.; Kumar, Y.; Brownlee, B. G. Chemosphere, 1978, 7, 155.
23. Ruzo, L.O.; Zabik, M. J.; Schuetz, R.D. J. Amer. Chem. Soc. 1974, 96, 3809.
24. Dulin, D.; Drossman, H.; Mill, T. Environ. Sci. Technol. 1986, 20, 72.
25. Miller, G. C.; Miille, M. J.; Crosby, D. G.; Sontum, S.; Zepp, R. G. Tetrahedron, 1979, 35, 1797.
26. Hustert, K,; Korte, F. Chemosphere 1974,3, 153.
27. Mansour, M.; Parlar, H.; Korte, F. Chemosphere 1980, 9, 59-60.
28. Ruzo,, L. O.; Sundstrom, G.; Hutzinger, O.; Safe, S. J. Royal Netherland Chem. Soc. 1977, 96, 249.
29. Bunce, N. J.; Bergsma, J.P.; Bergsma, M.D.; De Graaf, W.; Kumar, Y.; Ravanal, L. J. Org. Chem. 1980, 45, 3708.
30. Bunce, N. J.; Safe, S.; Ruzo, L. O. J. Chem. Soc. Perkin I 1975, 1607.
31. Chittim, B.; Safe, S.; Bunce, N. J.; Ruzo, L. O.; Olie, K.; Hutzinger, O. Can. J. Chem. 1978, 56, 1253.
32. Sundstrom, G.; Olie, K.; Hutzinger, O.; Safe, S.; Chittim, B. Chemosphere, 1977, 6, 103.
33. Ruzo, L.O.; Safe, S., Zabik, M. J. J. Agric. Food Chem. 1975, 23, 594.
34. Freeman, P. K.; Srinivasa, R. J. Agric. Food Chem. 1983, 31, 775.
35. Freeman, P. K.; Srinivasa, R. J. Agric. Food Chem. 1984, 32, 1313.
36. Freeman, P. K.; Jonas, V. J. Agric. Food Chem. 1984, 32, 1307.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch029

37. Bunce, N. J.; Pilon, P.; Ruzo, L. O.; Sturch, D. J. J. Org. Chem. 1976, 41, 3023.
38. Occhiucci, G.; Patacchiola, A. Chemosphere 1982, 11, 255.
39. Draper, W. M.; Crosby, D. G. J. Agric. Food Chem. 1983, 31, 734.
40. Bunce, N. J.; Kumar, Y.; Ravanal, L. J. Org. Chem. 1979, 44, 2612.
41. Ruzo, L. O.; Bunce, N. J. Tet. Lett. 1975, 511.
42. Ruzo, L. O.; Bunce, N. J.; Safe, S. Can. J. Chem. 1975, 53, 688.
43. Smothers, W. K.; Schanze, K. S.; Saltiel, J. J. Amer. Chem. Soc. 1979, 101, 1895.
44. Tsujimoto, K.; Tasaka, S.; Ohashi, M. J. Chem. Soc. Chem. Comm. 1975, 758.
45. Nishiwaki, T.; Usui, M.; Anda, K.; Hida, M. Bull. Chem. Soc. Jap. 1979, 52, 821.
46. Carey, J. H.; Lawrence, J.; Tosine, H. M. Bull. Environ. Contam. Toxicol. 1976, 16, 697.
47. Barbeni, M.; Pramauro, E.; Pelizzetti, E.; Borgarello, E.; Serpone, N. Chemosphere, 1985, 14, 195.
48. Rappe, C.; Nilsson, C. J. Chromatogr. 1972, 67, 247.
49. Nilsson, C.; Andersson, K.; Rappe, C.; Westermark, S. J. Chromatogr. 1974, 96, 137.
50. Choudhry, G. G.; Sundstrom, G.; Ruzo, L. O.; Hutzinger, O. J. Agric. Food Chem. 1977, 25, 1371.
51. Crosby, D. G.; Wong, A. S. Chemosphere 1976, 5, 327.
52. Crosby, D. G.; Wong, A. S.; Plimmer, J. R.; Klingebiel, U. I. Science (Washington, DC) 1971, 173, 748.
53. Crosby, D. G.; Wong, A. S. Science (Washington, DC) 1977, 195, 1337.
54. Hutzinger, O.; Blumich, M. J.; v. d. Berg, M.; Olie, K. Chemosphere 1985, 14, 581 and references cited therein.
55. Hutzinger, O.; Safe, S.; Wentzell, B. R.; Zitko, V. Environ. Health Perspect. 1973, 5, 267.
56. Mazer, T.; Hileman, F. D. Chemosphere 1982, 11, 651.
57. Safe, S.; Safe, L.; Mullin, M. J. Agric. Food Chem. 1985, 33, 24.
58. Safe, S.; Bandiera, S.; Sawyer, T.; Zmudzka, B.; Mason, G.; Romkes, M.; Denomme, M.; Sparling, J.; Okey, A.; Fujita, T. Environ. Health Perspect. 1985, 61, 21.
59. Leece, B.; Denomme, M. A.; Towner, R.; Li, S. M. A.; Safe, S. J. Toxicol. Environ. Health 1985, 16, 379.
60. Robertson, L. W.; Chittim, B.; Safe, S.; Mullin, M.; Pochini, C. M. J. Agric. Food Chem. 1983, 31, 454.

RECEIVED December 5, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch029

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch030

Chapter 30

Sampling and Decontamination Methods for Buildings Contaminated with Polychlorinated Dibenzodioxins

Richard L. Wade[1] and John P. Woodyard[2]

[1]International Technology Corporation, 4585 Pacheco Boulevard, Martinez, CA 94553
[2]International Technology Corporation, 23456 Hawthorne Boulevard, Torrance, CA 90505

The advent of state real estate transfer laws such as New Jersey's Environmental Cleanup Responsibility Act (ECRA), as well as Comprehensive Environmental Response, Compensation and Liability Act (CERCLA) and Resource Conservation and Recovery Act (RCRA) project evolution toward inclusion of buildings and equipment, has brought a new focus to technology for building and equipment decontamination. This paper reviews sampling and decontamination technology specifically for buildings and equipment contaminated with polychlorinated dibenzodioxins (PCDD). Several large-scale decontamination projects have been undertaken in which building structures, equipment, and machinery were decontaminated to remove PCDD and related compounds. The three basic types of building contamination are:

- Fugitive deposits of contamination from adjacent sites,
- Contamination from pyrolysis by-products generated during chemical fires, particularly PCB, and
- Incidental contact with contaminants during related product manufacture or storage (pentachlorophenol use, PCB electrical equipment, 2,4,5-T pesticide manufacture, etc.).

In any remediation effort, the selection of samples and implementation of sampling techniques is often considered as much an art as a science and depends heavily on the experience of the sampling team and knowledge of the former use of the building and/or transport mechanics for the contaminants. This paper focuses on the state of knowledge surrounding the decontamination of buildings and equipment and the associated sampling for project assessment and completion. The information contained here is derived primarily from project experience that is not documented in the literature.

0097-6156/87/0338-0367$06.00/0
© 1987 American Chemical Society

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch030

Sources of Contamination

In cases involving building and equipment decontamination, the first problem is to design and execute a sampling plan that reliably represents the degree and distribution of contamination. While it is often simple to define the cause of contamination and conceptually trace its path of transport, the task of defining the contamination perimeter is often made difficult by the redistribution of contaminants from tracking or airborne deposition. In particular, the following sources are possible routes:

- Direct deposit of airborne dust, soot, and fumes
- Indirect airborne distribution of contaminants by air circulation systems
- Direct mechanical contact through tracking by people and equipment
- Spreading of firefighting water or other fire suppressants used in the case of emergencies
- Careless use of other routine janitorial or industrial cleaning operations.

A thorough assessment of possible contamination therefore looks first at the logical sources, and then tracks possible cross-contamination through the routes mentioned above.

General Sampling Concepts

Sampling design must consider the physical nature of the contaminant source, its origin, and likely mechanisms for distribution. A thorough understanding of the mechanisms involved at each particular site is essential prior to initiating a full sampling protocol.

The first objective is to evaluate the contamination perimeter and its spread so as to effectively isolate contaminated areas from clean areas and prevent further spreading. This perimeter definition can be executed through a combination of a random sampling grid and a directed "pathway" analysis, the latter focusing on logical paths of mechanical tracking from the source. Until this perimeter is defined by the analytical results, all areas should be considered contaminated and isolated from the rest of the building.

Also, prior to finalizing a sampling plan, consideration should be given to the use of an analytical indicator, a compound that can be used to monitor the initial extent of contamination and the effectiveness of decontamination as the project proceeds. In the case of a PCB fire where PCDD and polychlorinated dibenzofurans (PCDF) are the contaminants of concern, PCB analysis costs approximately 10 percent of the cost of analysis for the other compounds. In most cases, a ratio between the two can be established and used to reduce the analytical cost and allow for a much broader sampling program.

Some representative ratios observed on PCB fire decontamination projects are shown in Table I. The ratio often changes due to selective removal in decontamination. Differences in vapor pressure and contaminant physical characteristics (soot, fume) can also affect the ratio.

Table I. Typical Range of PCB/PCDF Ratios During PCB Fire Decontamination

Medium/Stage	PCB/PCDF Ratio*
Surfaces	
• Pre-Decon (gross)	100 - 300
• Post-Decon	500 - 1,500
Air	5,000 - 10,000

* Total Polychlorinated Dibenzofurans

Analytical costs can be reduced significantly through the use of composites. In situations where large samples are required (1 m^2 for PCDD/PCDF), four 0.25 m^2 samples can be composited from a surface or an individual room. Composite sampling results of this type are often accepted by regulatory agencies in determining the effectiveness of cleanup.

Along with perimeter definition, additional data should be collected to evaluate the nature of contamination spread and its degree of penetration. Bulk samples of liquids, soils, and other dust should be collected and initially analyzed for PCDD/PCDF. Air samples can also be taken to demonstrate the variation in the ratios and determine the extent of contamination in air and resulting deposition on surfaces.

The development of a directed sampling program is relatively straightforward, involving selection of samples along routes where contamination is expected to be tracked or deposited. In addition, consideration is often given to the development of a grid system providing an overall contamination review for a facility. Such a grid system can be used for both random sampling plans as well as the three dimensional identification of sampling locations for the perimeter study.

The most commonly used grid systems are rectangular and triangular. The first critical decision in developing a grid system is to decide upon the desired sample number. As a guide, the sample number n can be determined from the following equation:

$$n = \frac{\text{Total Area (ft}^2\text{)}}{S^2(\text{ft}^2)},$$

where S is the distance between sampling locations or grid centers, determined by the size of the building (greater "S" for greater building size) and the perceived extent of contamination based on the nature of the incident.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch030

For triangular sampling grids, n is computed as follows:

$$n = \frac{0.66 \text{ X Total Area } (ft^2)}{T^2 \ (ft^2)}$$

where T is the distance between grid centers.

Once the grid is established, all samples can be obtained and analyzed, or a random selection scheme can be developed to sample only a subgroup, depending on budget. A common practice involves analyzing some randomly selected samples and archiving others for subsequent analysis if the results in a certain area prove positive. Another approach is to employ both the pathway and grid system, beginning with a pathway sampling design and employing the grid system once the pathway samples are demonstrated to be positive for PCDD or its indicators in a given area.

Sampling Plan Design

Three basic types of sampling programs are typically executed during a PCDD decontamination effort, in the following sequence:

- Perimeter sampling before decontamination
- Confirmatory sampling during decontamination
- Closure sampling following decontamination

Perimeter sampling identifies the extent of contamination. This phase of sampling is often the most expensive, as many of the samples are used to confirm that contamination does not exist, and identification of contamination hot zones often triggers additional sampling and analysis.

The nature of the contamination incident determines the sample types. Air samples and wipe samples are taken when dust or fume deposition is the problem. Core samples or chips may be taken from porous surfaces when contaminant penetration is expected to have occurred. The cores in particular are often sliced into sections, with analysis beginning in the top section or two and proceeding downward if results continue to prove positive. The results of the core/chip analysis determine the extent to which porous surface removal is required.

Confirmatory sampling is used by the decontamination crews to determine the effectiveness of the cleaning technique being employed. If a cleaned surface tests clean, the surface should be isolated from further airborne deposition.

Closure sampling may be the least expensive part of sampling and analysis on a project, but it is the most important. Decontamination standards for large projects are often set on the basis of an exposure assessment, which assumes an average contact between workers and contaminated surfaces. A sampling plan for closure takes this into account by scientifically selecting sampling locations that together provide an average contact surface for the subject worker. Sampling locations are therefore well defined in most cases and are typically not biased toward areas where contamination was known to exist. The end product of closure sampling includes averaging of the sampling results and a final risk assessment to determine whether the contaminant level is adequate based on the standard set by EPA, NIOSH, or the cognizant state agency.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch030

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch030

Table II describes some PCB fire incidents in recent years. The type of contamination varies between incidents based on the source of the problem (equipment overheating versus actual fire). Also, the levels of contamination, as computed as TCDD equivalents, vary significantly. Higher degrees of contamination often correspond with greater penetration into porous surfaces, and require different decontamination techniques and a more extensive three dimensional sampling program involving cores, chips, and bulk samples.

Quality control/quality assurance programs are critical to the success of decontamination efforts. For example, there are no concensus standard techniques for wipe sampling, although several variations on the same technique have been employed. The effectiveness of the sampling technique is not only a function of proper technique, but of the appropriateness of the chosen sampling method to detect contamination where it probably exists. In cases where fumes have deposited on painted surfaces, for example, repeated rewiping of the same location may continue to show contamination because the material has penetrated the paint and continues to diffuse to the surface. Knowledge of contaminant compatibility with surfaces combined with proper execution of the sampling technique is therefore important to the generation of useful data.

Decontamination Execution

A variety of decontamination techniques are used. Key considerations in the selection of techniques include the appropriateness of wet or dry application, the nature of contaminant deposition, the depth of contamination, the sensitivity of the project to decontamination or surface damage, and the availability of ratios to monitor effectiveness.

Three basic types of decontamination techniques exist:

- Surface techniques for removing surface deposits
- Removal techniques for penetrated contaminants
- Airborne removal techniques for soot and fume

The conceptual selection of techniques is straightforward, depending initially on the thoroughness of the perimeter sampling program. A list of the most commonly used techniques and their range of effectiveness are provided in Table III.

The selection of a technique depends on many factors. One major factor is the sensitivity of the particular location to cross contamination. Techniques such as sand blasting and hydroblasting are difficult to control, and, in areas where ultra clean levels are required, do not provide sufficient assurance that cross contamination does not occur. Wet techniques in particular, although commonly used as cleaning techniques (hydroblasting and pressure washing), are notorious for spreading contamination if not properly controlled. The sequence of cleaning with wet techniques in particular is important, as water settles toward the floor and recontaminates previously clean surfaces if walls are cleaned.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch030

Table II. Comparison of Noteworthy PCB Fire Incidents

Event	Date	Description	PCB/PCDF/PCDD Transport Soot	PCB/PCDF/PCDD Transport Fume	PCDF/PCB Ratio	Contamination Range 2,3,7,8-TCDD Equivalents (ng/m^2)	"Guidelines" 2,3,7,8-TCDD Equivalents (ng/m^2)
Reims, France	1985	Transformer Failure	No	Yes	1/110	> 124,000 (b)	-
Columbus, OH	1984	Capacitor Fire	Yes	Yes	1/200	> 20,000 (a)	20
Binghamton, NY	1981	Transformer Fire	Yes	Yes	1/1200	> 2,000 (b)	3.3-28
San Francisco, CA	1983	Transformer Fire	Yes	Yes	1/1000	> 1,300 (a)	3
Santa Fe, NM	1985	Transformer Fire	No	Yes	1/1000	> 20,000 (a)	1
Tulsa, OK	1984	Transformer Fire	No	Yes	1/2000	> 300 (a)	3.3-28
Montreal, Canada	1984	Capacitor	Yes	Yes	1/500	> 20,000 (a)	10

(a) Levels expressed in total PCDD/PCDF.
(b) Levels expressed in 2,3,7,8-TCDD equivalents using U.S. Environmental Protection Agency homologue method of calculation.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch030

Table III. Gross and Final Decontamination Techniques and Their Effectiveness

Contaminated Surface	Decon Technique	Removal Effectiveness % *
Concrete Floor +	• Scrubbing with detergent	50 - 95
	• Acid etching	80 - 98
	• Sandblasting/shotblasting	25 - 95
Unpainted Cinderblock Wall	• High pressure water spraying	60 - 90
	• Steam cleaning	90 - 99+
Galvanized Painted Ceiling and Walls	• High pressure water spraying	80 - 99+
Painted Steel Walls, Ceiling, Floor and Cabinets	• High pressure water spraying	70 - 95
	• Various methods	10 - 50
Electrical Equipment	• Solvent spraying	98 - 99

* Single Cleaning Pass
\+ Assuming short-term contact, no standing liquid

Dry techniques are used most commonly in commercial buildings. These techniques involve a light removal of surface deposits combined with vacuum attachments to minimize spreading of the cleaning media and the associated contaminants. Cleaning media include sand and grit, metal shot, granulated plastic, and less abrasive materials such as corn cobs and walnut shells. The potential for cross contamination from these media nevertheless exists; the choice is based primarily on the amount of damage that can be afforded on the clean surface. For some materials that are especially permeable or porous, decontamination is impractical. Disposal of materials such as fabric, wallpaper, wood, and other materials that are significantly contaminated is often the normal procedure.

Concrete presents a special problem. Because concrete is used as an integral structural component in many buildings, removal of concrete is not always appropriate or possible. Grinding and surface removal techniques are most often used for this application, and can be effective at up to two inches of depth. Few new developments in concrete decontamination have taken place. Most equipment suppliers and developers have focused either on increasing productivity or improving means of controlling fugitive dust emissions. In situ reagent-based techniques have been applied in Europe, but not in the United States. (1)

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch030

Decontamination Closure

Since there are no national standards for decontamination, each site requires a specific exposure assessment to determine the appropriate "safe level" of PCDD contamination. The exposure assessment is normally developed as part of the remediation plan and is submitted with the closure sampling plan to the cognizant regulatory agency before beginning work.

Most closure sampling efforts therefore focus more on the overall level of contamination remaining. In accordance with the exposure assessment, average contamination levels need to be determined in order to qualify any long-term exposure risk. Proper closure sampling normally includes a good, statistically sound sample selection system that generates high and low contact contamination estimates by work zone. Use of such a statistical approach prevents disputes about cleanliness based on one or two positive samples.

The end result of closure sampling is a series of contamination statistics for different contact surfaces. These data and the exposure model determine the exposure risk to workers.

Conclusion

Building decontamination technology continues to advance as each project is executed. New technologies are developed for specific applications, and participants gain a better understanding of the importance of exposure assessments, closure sampling, and

perimeter sampling to successful completion of a project. A better understanding of decontamination techniques is needed, particularly for concrete and porous materials that may not be expendable as part of the decontamination program. Refinements in concrete removal technology and in situ treatment are expected to dramatically improve the state of the art within the next several years.

An improved understanding of closure sampling is an important part of the decontamination process, one which is unfortunately limited to only those state, NIOSH and EPA regional offices with extended experience in decontamination projects. The advent of national decontamination standards for PCDD, PCB, and other organic contaminants may not be the answer; each site is cleaned to a level that does not present an unreasonable risk to human health, and the considerations involved in that determination vary from site to site. It is therefore important that regulatory agencies gain a better understanding of proper closure sampling, and that a concensus be developed as to what constitutes a complete closure sampling plan.

Literature Cited

1. Tumiatti, W.; Nobile, G.; Tundo, P.; Division of Environmental Chemistry Preprints, American Chemical Society 191st National Meeting, New York, 1986, p.259.

RECEIVED January 21, 1987

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch030

Chapter 31

Chemical Degradation of Selected Polychlorinated Compounds by Means of Polyethers, a Base, and an Oxidant

G. Nobile[1], W. Tumiatti[1], and P. Tundo[2]

[1]Sea Marconi Technologies, Strada Antica di Collegno, 196-10146 Torino, Italy
[2]Istituto di Chimica Organica dell'Università di Torino, Via G. Bidone, 36-10125 Torino, Italy

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch031

Mixtures of polyethyleneglycols (and ethylene oxide-propylene oxide copolymers), a base and a peroxide (or other radical initiators) allow the preparation of several reagents which, suitably formulated according to their different use, are able to degrade the chemically stable chlorinated aromatics. Such a method, called CDP-Process, is active on TCDD and can be applied in different ways; as an example, the reagent, when immobilized on a solid bed, allows the continuous-flow decontamination of mineral oils containing PCB; this is useful for the decontamination of an electrical transformer during operation. Another reported example is the decontamination of surfaces contaminated by PCB or PCB fires (where PCDF and PCDD are also present).

Since the ecological and environmental hazard related to stable polyhalogenated aromatic compounds has been recognized, many efforts have been done by chemists both for limiting their spreading in the environment and for their elimination. In order to reach this aim the contaminated material is put in landfills or incinerated, or the organic pollutants are chemically decomposed. Each method has its peculiarity and applicability. The advantages of chemical methods of decontamination are in the ultimate elimination of the problem, in the possibility of operating in situ, and in the use of inexpensive equipment. For these reasons new chemical methods able to destroy the stable molecules of chlorinated aromatics are being examined.

For polychorinated biphenyls (PCB), many methods have been reported. For example: the reaction of polyethyleneglycols (PEG) of low molecular weight (MW) in the presence of strong bases (i.e. KOH) and stirring (1, 2); the use of sodium polyethyleneglycolates (3); the utilization of alkali metal hydroxide/glycol mixture in the presence of dimethylsulfoxide (4) and u.v. irradiation (5). Several methods have also been described for the more toxic of such compounds, that is the 2,3,7,8- tetrachlorodibenzo-p-dioxin (TCDD); among them, remarkable are the u.v. irradiation (6), the

0097-6156/87/0338-0376$06.00/0
© 1987 American Chemical Society

exhaustive chlorination (7), the reaction with sodium glycolates (8).

A new method of degradation, called CDP-Process (Chemical Degradation of Polyhalogenated compounds), is described together with its application in the field (9). According to the CDP-Process polychlorinated compounds are easily degraded if the reaction is carried out in the presence of PEG of high MW, a base (even K_2 CO_3 or $NaHCO_3$ can be used), a peroxide or a radical initiator; moreover, the reagents mixture can be suitably formulated in order to obtain different applications.

CDP-Process

Application of this method to TCDD, has been previously reported (10). The related experiments were performed at the Establishment of the European Community (Ispra) and at the ICMESA plant (Seveso); these reactions, carried out on pure dioxin and on several contaminated materials of the reactor, gave good results (11).

In Figure 1 is shown the elimination of pure TCDD from a n-decane solution. In both cases the two immiscible phases were stirred magnetically. Analyses were carried out on the hydrocarbon solution. The sole identifiable products in the reaction mixture were dibenzodioxins progressively dechlorinated (no cine substitution was observed); moreover, no trace of compounds containing - OH in place of - Cl was detected (GC/MS). Attention was not addressed in detection of eventual compounds having a polyethyleneglycol group bonded to the aromatic, since their chemical properties, very close to those of the reagent, make their separation very difficult. So, no quantitative results are at the moment available. It was also reported that the reaction mixture was not toxic on the guinea pigs (10).

Such results indicate that, from a mechanistic point of view, it is not a matter of a simple aromatic nucleophilic substitution (in any case very difficult if not via benzyne), but more probably of a radical substitution on an activated complex of the TCDD (SRN1 mechanism) (12); this mechanism is favoured by the fact that the radical anion of TCDD is particularly stabilized (13). How this radical anion is generated from the initiator peroxide and how it reacts with the hydrogen donor (PEG or the solvent) is not at this moment clear; however, interesting to note, degradation of TCDD by u.v. irradiation leads to the same intermediate compounds.

The difference occurring in reactions carried out in the presence of peroxide or of strong bases (1-4) is explained by the following example which moreover illustrates a decontamination from PCB of mineral oils:

5.0 mL of mineral oil contaminated with 1300 ppm of PCB (Arochlor 1260) were magnetically stirred at 80 °C with 2.5 g of two different mixtures (A and B; % are in weight). A: 86% PEG 6000 (PEG of MW = 6000), 12% K_2CO_3, 2% Na_2O_2. B: 77.5% PEG 6000, 10% K_2CO_3, 12.5% CH_3ONa. Gas chromatographic analyses on the oil showed the following residual contaminations (ppm of PCB after 0.5, 1.0 and 1.5 h, respectively); A: 90, 26 and 13; B: 350, 211 and 142. From the respective concentration ratios it results that the reaction carried out with sodium peroxide is at least 15 times faster than

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch031

the reaction carried out with methoxide, in spite of the stronger base character of the latter; accordingly, the reaction is not related to the strength of the base although its presence is needed (sodium bicarbonate is also effective if a peroxide is present). Other data indicate that the reaction does not depend on stirring; it is possible to operate with other peroxides (BaO_2 as an example) and even in the presence of water (till 30% is allowed).

Although the reaction mechanism is not at the moment fully clarified, some points seem to be well established: the radical character of the reaction and the contemporary need of an anion activator as PEG is (maybe the function of the anion activator is to increase the strength of the base as they do in Phase-Transfer Catalysis) (14). In place of PEG other anion activators may be used, as the condensation compounds between ethylene oxide and propylene oxide; such latter polymers, being viscous liquids, offer some advantage in particular applications such as the decontamination of surfaces.

Applications

The CDP-Process works well on several stable, halogenated aromatic derivatives; mainly studied, also in connection with its industrial application has been the PCB and the degradation products that PCB forms as a consequence of fire; that is polychlorinated dibenzofurans and dioxins (PCDF and PCDD, respectively).

By suitably changing the type of polyether, the base and the peroxide, and in case by adding a solvent, different "reagents" can be prepared which are able to attack and decompose such supertoxic molecules even if they are present in different materials (mineral oils, surfaces, soil, etc.); in fact different procedures must be followed, in connection with the type of pollutant, the environmental requirements, and the involved materials.

A few applications of the method that illustrate its peculiarities are shown.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch031

Decontamination of Mineral Oil Containing PCB in a Batch Process. Table I reports some results of CDP-Process in destroying PCB from its hydrocarbon solutions (for analytical convenience an n-decane solution was used). The reaction is effective even in the presence of actually catalytic quantities of peroxide; without peroxide, no reaction occurs at all.

At the end of the reactions no trace of PEG was detected in n-decane (experiments of Table 1) or in mineral oils treated in the same way (IR and NMR analysis). In the reactions of Table 1 as in the following ones here reported, the residual PCB were usually the low-chlorinated polychlorobiphenyls; these ones have been reported to be the less toxic.

The electrical properties of mineral oils after the CDP-Process render such dielectric fluids suitable for their use in electrical transformer without further treatment (usually the dielectrical characteristics of the oil are improved).

Table I. Chemical Degradation of PCB by CDP-Process. Influence of % Na_2O_2

% Na_2O_2 in the reagent mixture	Initial Contamination (ppm)	Final Contamination (ppm)
8	16,000	23
2	16,000	32
0.78	16,000	190
0.21	16,000	5000
0.00	16,000	no reaction

Conditions: 3.0 mL of n-decane, 2.0 g of reagent mixture: 12% (by weight) K_2CO_3 and the difference at 100 of PEG 6000; T = 85°C; magnetic stirring for 3 hours.

Decontamination of Mineral Oils Containing PCB, in an Open and a Closed Continuous-Flow Process. The reagents in the CDP-Process can be immobilized by adsorption onto a soild bed; in such a way a solid reagent can be produced which can be used to decontaminate PCB mineral oils passing through it (15). This is made possible, from a chemical point of view, by two facts: i. the reagent is not soluble in the oil. ii. the PCB's decomposition products are completely retained on the solid bed (the PCB are not; it is not a matter of adsorbtion). Figure 2 illustrates the results obtained working with an immobilized reagent and by flowing through the contaminated oil; the oil containing 900 ppm PCB flowed continously into the column containing a bed of 30 g solid reagent (2.6 g PEG 6000, 0.7 g potassium t-butoxide, 0.7 g Na_2O_2, 26 g K_2CO_3) and was collected at its outlet. As shown in Figure 2, the column is progressively exhausted; operating in appropriate conditions and changing the reactive bed when it becomes exhausted or operating in series with more than one column, one can obtain a decontaminated oil without the troubles of batch operation (stirring, charge of the reactor, drain, incidental filtration or centrifugation of the oil, etc.).

Another version of the CDP-Process utilizing immobilized reagents is a continuous and closed process; accordingly, the same contaminated oil is continuously recycled in the same column until the decontamination limits imposed by law are reached. The commercial version of such continuous processes involves the presence of the immobilized reagent in cartridges, easy to handle and to transport; such cartridges of standard dimensions are the core of a plant which continuously pumps the contaminated oil from a tank, heats it to the desired temperature, sends it to the cartridge and gives it back to the tank. In this way the volume of the oil to be treated is independent from the volume of the plant: it is enough to change the cartridge when it becomes exhuasted.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch031

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch031

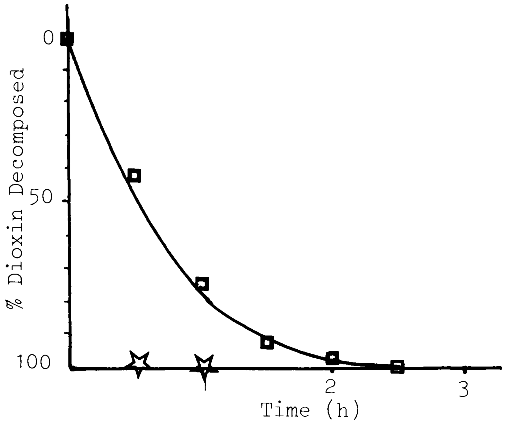


Fig. 1. Degradation of TCDD.
• T = 50°C; 2.0 mL of n-decane containing 5 g of TCDD, 0.9 g PEG 1500, 0.15 g K_2CO_3, 0.10 g diethyleneglycolmonobuthylether, 0.10 g Na_2O_2.
• T = 85°C; 2.0 mL of n-decane containing 5 g of TCDD, 2.06 g PEG 6000, 0.53 g K_2CO_3 and 0.37 g Na_2O_2.

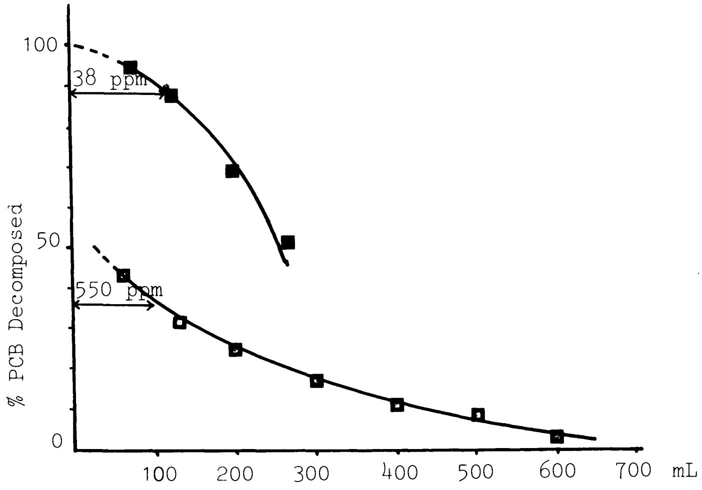


Fig. 2. Decontamination from PCB of Mineral Oil in a Continuous and Open Process.
• T = 90°C; flow 60 mL/h (the first 120 mL contained 38 ppm of PCB).
• T = 60°C; flow 120 mL/h (the first 100 mL contained 550 ppm of PCB).

Figure 3 shows how the PCB contamination of a large quantity of oil is progressively decreased to 42 ppm by changing five cartridges containing 16 Kg each of solid bed. In this experiment, 440 kg of oil containing 4,000 ppm PCB was recycled continuously at 100 L/h at 80° C.

Some advantages of such a version of the method are that the plant, which can be closed in a container, works without the steady presence of manpower and that it can be directly connected to the apparatus containing the oil to be decontaminated. Such characteristics allow this continuous-flow method, its reagents and conditions to be employed in the decontamination of electrical transformers.

Decontamination of Surfaces Contaminated by PCB, PCDF and PCDD. This version of CDP-Process uses reagent mixtures in liquid phase and at room temperature; the reagent is spread on the surface to be decontaminated. Usually the reagent is formulated by using copolymers between ethylene oxide and propylene oxide (these condensation polymers, manufactured by Montedison, are called 'Nixolen').

The efficacy of the reaction was peviously reported on TCDD (10). Here is described an application in the field, for the clean-up of surfaces contaminated by PCDF and PCDD coming from a PCB accident.

In an electrical sub-station in Italy, because of a short-circuit, a rectifier with about 300 little capacitors containing PCB burned (50 g each capacitor and a total amount of PCB of about 10 Kg). The fire lasted for about one hour before it was extinguished. The thermal oxidation of PCB produced a large quantity of soot containing PCDF and PCDD. The soot was spread inside the sub-station contaminating about 1500 m^2 of surfaces and items.

During the sampling operations for the mapping of the contamination and the data acquisition necessary for the design of the decontamination, a few tests were performed to characterize the best method of application of CDP-Process in order to reach the complete in situ destruction of PCDF and PCDD.

Different surfaces (floor, roof, walls) were selected to perform the assays. On those surfaces the reagent, liquid at room temperature (95% of Nixolen and 5% of sodium alkoxide), was spread at room temperature to produce a liquid film; such a film at the same time avoided the transfer of the contaminant to the environment. After about 90 hours of action at room temperature, the liquid film was then removed. Samples were taken either on the surfaces (wipe tests, before and after the treatment were made) and on the removed reagent.

Table 2 shows the values of contamination on these representative surfaces before and the results after decontamination. No contaminant was detected in the removed reagent. All these data demonstrate the effective degradation of PCDF and PCDD by CDP-Process, either on the surfaces and in the reagent.

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch031

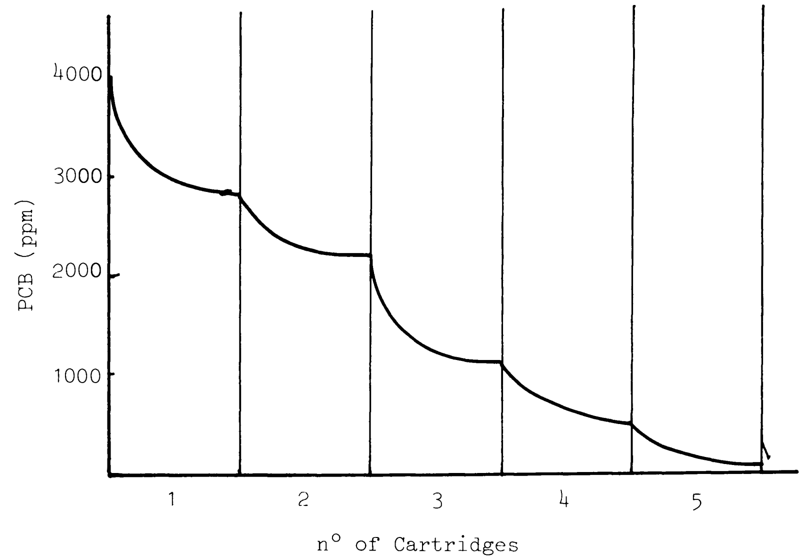


Fig. 3. Decontamination from PCB of Mineral Oil in a Continuous and Closed Process.

Table II. In Situ Decontamination of Surfaces Containing PCDF and PCDD, Coming from a PCB Fire

Contaminant (PCDF + PCDD)	Sample Taken on Floor Before (ng/m^2)	After (ng/m^2)	Sample Taken on Roof Before (ng/m^2)	After (ng/m^2)
2,3,7,8-TCDF	256	ND	1060	174
TCDF	1169	27	4841	362
PeCDF	678	13	2809	92
HxCDF	426	ND	1762	22
HpCDF	336	11	1391	ND
OCDF	284	20	1029	ND
2,3,7,8-TCDD	ND	ND	33	ND
TCDD	24	ND	100	ND
PeCDD	36	ND	148	ND
HxCDD	45	ND	187	ND
HpCDD	42	ND	174	ND
OCDD	35	114	143	ND

ND = Not Detectable (detection limit depends from the sample, varying from 2 to 8 ng/m^2).

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch031

Acknowledgments

This work was partly a research program of the Consiglio Nazionale delle Ricerche, Progetto Finalizzato 'Chimica Fine e Secondaria'.

Literature Cited

1. Brunelle, D. J.; Singleton, D.A. Chemosphere 1983, 12, 183.
2. Brunelle, D. J. U.S. Patent Appl. n° 314,163 (1981) (General Electric Company).
3. Pytlewsky, L. L.; Krevitz, K.; Smith, A. B. U.S. Patent Appl. n° 142,865 and 158,359 (1980) (Franklin Institute).
4. Peterson, R. L. U.S. Patent Appl. n° 501,620 (1983) (Galson Research Corporation).
5. Draper, W. M. Chemosphere, 1985, 14, 1195.
6. Kitchens, J. A. F. U.S. Patent Appl. n° 890,871 (Atlantic Research Corporation).
7. Shannahan, C. E. et al. Report EPA-600/-78-146 (1978).
8. Howard, K. J.; Sidwell, A. E. Eur. Patent n° 0 021 294 A1 (Vertac Chem. Corporation).
9. Tundo, P. U.S. Patent Appl. n° 771,404 (1985) (Sea Marconi Technologies).
10. Tundo, P.; Facchetti, S.; Tumiatti W.; Fortunati, U. Chemosphere, 1985, 14, 403.
11. Tundo, P.; Facchetti, S.; Tumiatti, W.; Fortunati, U. Report to the President of the Lombardia Region (1983).
12. March, J. "Advanced Organic Chemistry"; Wiley: New York, 1985; 3rd Ed.; p. 582.
13. Miller, G.; Sontum, S.; Crosby, G. Bull. Environ. Contam. Toxicol. 1977, 18, 611.
14. Starks, C. M.; Liotta, C. "Phase Transfer Catalysis"; Academic Press: New York, 1978.
15. Tundo, P. U.S. Patent Appl. n° 632,718 (1984) (Sea Marconi Technologies).

RECEIVED December 5, 1986

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ch031

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ix001

Author Index

Alperin, E., 319
Aust, Steven D., 340
Babb, Tony, 267
Berridge, Bernard, 105
Boyd, Jimmy, 299
Bridges, J., 332
Brunck, Robert, 105
Bryan, R. C., 246
Bumpus, John A., 340
Carasea, Angelo, 259
Carson, C. P., Jr., 278
Casanova, Judy N., 229
Condorelli, P., 332
Daily, Philip L., 311
Dei Rossi, Dale, 286
des Rosiers, Paul E., 34
Dev, H., 332
Downey, D., 332
Draper, William M., 350
Exner, Jurgen H., 1
Fingerhut, M. A., 142
Forrester, Ray K., 278
Fox, R., 319
Freeman, Raymond A., 114
Gallo, Michael A., 131
Gasiewicz, Thomas A., 162
Geisler, T., 319
Gerken, Mary, 259
Groen, A., 319
Hallett, Douglas J., 94
Halperin, W. E., 142
Hazel, Ralph, 216
Helsel, R., 319
Hesse, Elizabeth J., 131
Hileman, Frederick D., 114
Houk, Vernon N., 68,174
Jurgens, H.-J., 221
Kay, Morris, 216
Kimbrough, Renate D., 68
Kleopfer, Robert, 259
Kociba, R. J., 54
Marple, Leland, 105,278,286
Milicic, E., 291
Miller, Glenn C., 82
Morey, Debra, 259
Murray, F. J., 178
Nobile, G., 376
Noble, Roy W., 114
Novosad, C., 291
Olfenbuttel, Robert F., 229
Paustenbach, D. J., 178
Peterson, R., 291
Rappe, Christoffer, 20
Rogers, C., 291,332
Roth, R., 221
Ruzo, Luis O., 350
Schecter, Arnold, 162
Schnorr, T. M., 142
Schroy, Jerry M., 114
Shu, H. P., 178
Splitstone, D. E., 246
Stephens, Robert D., 350
Stoddart, T. L., 299,319
Sweeney, M. H., 142
Thomas, R. W., 299
Throop, Lewis, 105,286
Tumiatti, W., 376
Tundo, P., 376
Umbreit, Thomas H., 131
Wade, Richard L., 367
Williams, H. D., 299,319
Woodyard, John P., 367
Zepp, Richard G., 82

Affiliation Index

California Department of Health Services, 350
Carson-Mitchell, Inc., 278
Centers for Disease Control, 68,174
Dekonta GmbH, 221
Dow Chemical Company, 54
EG&G Idaho, 229,299,319
ELI--Eco Logic Inc., 94
Galson Research Corporation, 291
Geostat Systems, Inc., 246
IIT Research Institute, 332
International Technology Corporation, 1,246,267,319,367
J. M. Huber Corporation, 299
Michigan State University, 340
Monsanto Company, 114
National Institute for Occupational Safety and Health, 142
Sea Marconi Technologies, 376

In Solving Hazardous Waste Problems; Exner, J.;
ACS Symposium Series; American Chemical Society: Washington, DC, 1987.

Shirco Infrared Systems, Inc., 311
State University of New York, 162
Syntex (USA), Inc., 105,178,278,286
U.S. Air Force, 229,299,319,332
U.S. Department of Health and Human Services, 68,174
U.S. Environmental Protection Agency, 34,82,216,259,291,332
Universita di Torino, 376
University of California--Berkeley, 350
University of Medicine and Dentistry of New Jersey, 131
University of Minnesota, 350
University of Nevada--Reno, 82
University of Rochester Medical Center, 162
University of Umeå, 20

Subject Index

A

Abiotic transformation, 2,3,7,8-TCDD, 86-90
Absorption spectra, 2,3,7,8-TCDD, 86
Acceptable daily intake
  safety factor approach, 196
  2,3,7,8-TCDD, 163
Acute lethality, comparative toxicity of dioxins, 55
Adipose tissue
  chemical residues, 100t
  PCDD and PCDF levels, 27-28
  2,3,7,8-TCDD levels, 76,164
Adults, uptake of contaminated soil, 184
Advanced electric reactor
  destruction of dioxin contamination, 299-309
  diagram, 301f
  full-scale operation, 308
  method, 300-306
  particle production, 307
  personnel protection equipment, 304
  postreactor treatment, 302
  process diagram, 303f
  process parameters, 304t
  site conditions, 302
Advanced electric reactor feedstock, inorganic concentrations, 307t
Agent Orange
  active ingredients, 230
  association with birth defects, 153
  composition, 299
  incineration at sea, 230
  photolysis on glass surfaces, 88
  soft tissue sarcomas, 151-152
  soil decontamination, 319-331
Air Force Engineering and Services Center, research and development program, 229
Air monitoring survey
  analytical methods, 270
  basic steps, 269
  data management, 273
  design, 268-269
Air monitoring survey--Continued
  information needed, 268
  objectives, 269
  parameters, 269
  quality control, 273
  sampling
    duration, 270
    method validation, 273
    site selection, 269-270
  See also Ambient air monitoring
Alewives, role in ecosystem, 99
Alum flocculation process, removal of dioxin-bearing particulates, 280
Aluminum, needed for flocculation, 287
Ambient air monitoring
  description, 267-276,305
  See also Air monitoring survey
American Society of Mechanical Engineers (ASME), 44
Ames test, mutagenicity studies, 196
Analytical methodology
  air monitoring survey, 270
  2,3,7,8-TCDD, 259-265
Analytical procedures, hazardous waste cleanup, 263-264
Analytical support, 2,3,7,8-TCDD-contaminated sites, 259-265
Animals
  ingestion of soil, 205-206
  PCDDs, 7
  species susceptibility to dioxin, 175
  toxicity data, deriving exposure control limit for humans, 62-65
Arkansas
  dioxin cleanup, 13-14
  2,3,7,8-TCDD contamination, 35
Aroclor 1254, photodecomposition, 356f
Aroclor 1260, photochemical decomposition on concrete, 361f
Aroclor mixtures
  mineralized by *P. chrysosporium*, 347
  See also Polychlorinated biphenyls (PCBs)

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ix002

Aromatic halides, toxicity testing, 292
Aryl hydrocarbon hydroxylase (AHH)
  determinations in rat liver microsomes, 136
  dose-response relationship with 2,3,7,8-TCDD, 134
  enzyme induction, correlation with feeding studies in rats, 61t
  in rats, 61t,137t,187
  induction, 2,3,7,8-TCDD required, 138
Automobiles, source of PCDDs and PCDFs, 23
Average daily intake, dioxins by nursing infant, 168-171

B

Backfill, dioxin-contaminated lagoon, 285
Baltic Sea, PCDDs and PCDFs, 26t
Bedrock wells, 279
Benching
  description, 279
  dioxin-contaminated site, 282-283
Bernoulli distribution, mean and variance, 251
Bioavailability
  calculation methods, 188
  definition, 186
  dermal absorption, 186-187
  ingestion, 187-189
  inhalation, 189
  role of organic matter, 188
  2,3,7,8-TCDD from contaminated soils, 131-138
  2,3,7,8-TCDD in guinea pigs, 134
  variability, 132
Biodegradation, chlorinated organic compounds, 340-348
Biological degradations, dioxin, 6
Biomagnifications, PCBs, 351
Blepharitis, 70
Bound-wave exciters, design, 336
Breast milk
  chemical residues, 100t
  PCDD and PCDF levels, 28
Buildings
  contamination
    sources, 367-368
    techniques, 374
    types, 367
  decontamination, 226-227,367-375

C

Canada, PCDDs and PCDFs in humans, 165t
Cancer, role of 2,3,7,8-TCDD, 65
Carcinogenesis, promotion versus initiation, 194-195
Case-control studies
  design description, 147
  phenoxy herbicides and chlorophenols, 150,151t
Catalytic converter, PCDDs and PCDFs, 23
Cattle
  soil ingestion, 206
  2,3,7,8-TCDD analyses in Seveso, 27t
CDP process
  applications, 378
  degradation of PCB, 379t
Census data, epidemiologic studies, 152-153
Center for Environmental Health, 174
Centers for Disease Control (CDC)
  applications of methodology, 191
  assessment, 208
  dermal contact with dioxin, 186
  exposure assumptions, 192t-193t
  risk assessment, 179,193-194
  soil dioxin limit, 300
  study of Vietnam veterans, 77
CERCLA--See Comprehensive Environmental Response, Compensation, and Liability Act, Superfund
Chemical degradation of polyhalogenated compounds, process description, 377-378
Chemical workers
  cohort studies, 149t
  medical studies, 155t
Chloracne
  description, 143
  duration, 70
  persistence, 158
  symptoms of dioxin, 7
Chlordane, mineralization, 345t
Chlorinated compounds, degradation by *P. chrysosporium*, 344-346
Chlorinated dibenzodioxins
  human health effects, 68-77
  photolysis tests, 329
  thermal desorption tests, 328
  UV stability, 363
Chlorinated dibenzofurans
  photolysis tests, 329
  thermal desorption tests, 328
Chlorinated terphenyls, photolysis, 360
Chloronaphthalenes, photolysis, 360
Chlorophenols, associations with exposure, 158
Cinnamyl alcohols, free radical oxidative polymerization, 341
Clean Water Act of 1977, 231
Cleanup
  dioxin-contaminated site, 217,225,281-284
  evaluation of need, 192

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ix002

Closure sampling
description, 370-371
importance, 375
Coagulation, dioxin-contaminated sludge, 287
Cohort studies
chemical workers, 148,149t
description, 147
herbicide sprayers, 150t
Combustion air, portable infrared incinerator, 314
Combustion processes, chlorinated dioxins, 84
Composite sampling, 369
Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA)
description, 367
See also Superfund
Concentration, 2,3,7,8-TCDD in plants, 128
Concrete, decontamination, 357,375
Confirmatory sampling, description, 370
Conjunctivitis, 70
Consumer Product Safety Act, 231
Contamination
mappings, 247
perimeter definition, 368
sources, 368
suspended particle relationship, 275
See also Buildings, Cross contamination, Dioxin
Contract laboratory mechanism, 260-263
Copper smelters, PCDD and PCDF source, 22
Coronary vascular disease, association with 2,4,5-T, 154
Cost, dioxin cleanup, 179
Crane pad, dioxin-contaminated site, 281
Crops, dioxin uptake, 205
Cross contamination, 371
Cross-sectional studies, 147,154-158
Crosswalk software, 263
Crustaceans, PCDDs and PCDFs, 26-27

D

Data acquisition, portable infrared incinerator, 315
Data handling, contract laboratory program procedure, 262-263
Data management, ambient air monitoring survey, 273
dBase III, 262
Dechlorination
P. chrysosporium, 346
PCBs, 358-360
Decontamination
advantages of chemical methods, 376
buildings, 367-375
closure, 374
concrete, 374
dioxins, 291
herbicide plant, 221-228
quality assurance programs, 371
techniques, 371,373t
See also Dioxin
Delisting
change required, 239
concern, 239
contaminated soil, 236
decontaminated soil, 237-238
Department of Defense, 230
Dermal contact, dioxin exposure, 185
Descriptive medical reports, dioxin exposure, 143-146
Desorption, testing, 322-323
Detoxification, capability of the advanced electric reactor, 305
Dewatering, dioxin-contaminated site, 282
Dibenzofurans, human breast milk levels, 162-172
Dichlorodiphenyltrichloroethane (DDT), 100,248
2,5-Dichlorophenol, thermal decomposition, 221
Dielectric heating, mechanism, 335
Dielectric parameters, use, 336
Dielectric properties, soil, 335
Digestive system disease, association with 2,4,5-T, 154
Dimethyl sulfoxide, 292
Dioxin
acute medical consequences, 158
advanced electric reactor, destruction ability, 306
analysis, tandem mass spectrometry procedure, 261-262
basis of concern, 35
behavior in conventional water treatment, 286
binding to soil, 175-176
cleanup of herbicide plant, 225
combustion products, 300
contamination
analytical requirements, 260
cleanup, 218-219,236
community relations, 217-219
destruction, 299-309
permanent solutions, 219
plan development, 279
storage, 280
surface impoundment remediation, 278-285
temporary measures, 217
water treatment process, 280
dechlorination, 292
decomposition, 324

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ix002

Dioxin--Continued
decontamination from soils, 291-298
distribution of tier 1 and 2 sites, 39f
dose-response relationship, 197,199
effect of long-term exposure, 175
effect of weather on soil distribution, 298
effect on human health, 2-5
environmental entry, 267-268
environmental extent, 4
epidemiology, 142-158
exposure
dermal contact, 185-186
history, 144f
inhalation, 185
routes, 6,175
soil ingestion, 181-185
extraction from soil macropores, 296
factors that influence bioavailability, 186-187
flocculation with aluminum salts, 288t
genotoxicity, 195
hazardous waste problems, 1-16
health hazards of contaminated soil, 178-208
hepatotoxic effects, 145
high-volume sampler
effectiveness, 273
results, 274t
highly toxic members, 131
humans
breast milk levels, 162-172
health effects, 7
industrial explosions, 145t
isomers, 142
land disposal, 52
leaching into groundwater, 14
legal obstacles to cleanup, 10
mechanism of action, 195
microbial destruction, 14
migration, 6
neurologic effects, 145
no-observable effect level, 196
octanol-water partition coefficient, 108t,110t
pathway between ecosystems, 96
production, 37,142
removal
flocculation, 289t
from tap water, 289t
modified alum flocculations, 290
research program, 241
sample screening techniques, 274-276
sampled tier 3 sites, 43f
site-specific factors in remediation, 13t
sites
residential versus industrial, 189-191
United States, 40

Dioxin--Continued
sorption particles, 85
storage, structure decontamination, 219
thermal destruction, 237-238,291,311-318
uncertainties in risk assessment, 174-177
variation of octanol-water partition coefficients, 108t
virtually safe dose, 195
waste incineration, 14-15
water solubility, 106t,107t
See also 2,3,7,8-Tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD)
Dioxin Disposal Advisory Group, 240
Dirt, adult ingestion, 184
DNA, role in tumor growth, 194
Dog, acute toxicity of 2,3,7,8-TCDD, 5t
Domestic animals
dioxin contamination, 207
dioxin exposure, 205-207
Dose-response curve
dioxin, 176,199
extrapolation to low doses, 197
Dust, deposition in rural environments, 184

E

Effluent limits, dioxin in herbicide plant, 224
Eglin Air Force Base
Agent Orange use, 300
PCDDs, 85
thermal desorption testing, 322-323
See also U.S. Air Force
Electric energy, heating earth formations, 336
Electrical arcing furnaces, PCDD and PCDF sources, 22
Electromagnetic energy
conversion to heat, 332
depth of penetration, 335
Environmental Cleanup Responsibility Act, 367
Environmental laws, basic goal, 230
Environmental Protection Agency (EPA)
ban on PCB manufacture, 351
cleanup technology, 216
dermal contact with dioxin, 186
development and recommendations on research demonstration projects, 242
dioxin contamination limit, 300
guidance on delisting, 239
hazardous characteristics, 307
high-volume sampler, 270
national dioxin strategy, 35-36

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ix002

EPA--Continued
risk assessment models, 195
2,3,7,8-TCDD designation, 246
Environmental samples, levels of PCDDs and PCDFs, 23-27
Epidemiology
choice of study design, 146-147
current studies, 157f
definition, 146
dioxin, 142-158
Equipment
decontamination, 226-227
target levels for decontamination, 228t
Erosion, dioxin migration, 6
Excavation sequence, dioxin-contaminated site, 283-284
Exhaust system, portable infrared incinerator, 315
Exposure
assumptions in residential cleanup site, 192t
definition, 148
limits, 8
Extinction coefficients, PCBs, 359t
Extraction procedure toxicity, advanced electric reactor, 307t

F

Federal Emergency Management Agency, 37,217
Federal Insecticide, Fungicide, and Rodenticide Act, 231
Federal Register, 239
Federal Water Pollution Control Act, 230
Feed preparation system, portable infrared incinerator, 312
Field testing, soil decontamination, 295-296
Fish, 2,3,7,8-TCDD contamination, 50
Flocculation
dioxin-contaminated water, 287
removal of chlorinated organic compounds, 288
Fly ash, PCDDs and PCDFs in samples, 22
Food, route of exposure, 99-100
Food and Drug Administration (FDA), 37,62,195,196
Fringing-field line, 337,338f
Furans
advanced electric reactor, destruction ability, 306
decomposition from UV light, 324
highly toxic members, 131

G

Gas chromatography-mass spectrometry
analyses in mobility study, 119-120
Gas chromatography-mass spectrometry --Continued
2,3,7,8-TCDD analysis, 261
Genotoxicity, dioxin, 195
Geometry, PCBs, 358
Geophasia, incidence, 182
Geostatistics, basic concept, 247
Germany, herbicide plant closing, 221-228
Goats, 2,3,7,8-TCDD analyses in Seveso, 27t
Great Lakes
contamination of fish, 50
ecosystem, 95-99
PCDDs and PCDFs, 25-26
surface-to-volume ratio, 95
Greenpeace, 224
Grid systems, sampling, 369-370
Groundwater, dioxin contamination from herbicide plant, 225-226
Guinea pigs, 2,3,7,8-TCDD toxicity, 5t,55,69,134

H

Half-life, PCDDs, 85
Haloarenes, photoreduction scheme, 356f
Haloaromatic compounds, photolysis, 360
Hamburg, Germany, effect of dioxin, 3
Hamster, 2,3,7,8-TCDD toxicity, 5t,55,68-69
Hazardous and Solid Waste Act of 1984, 48
Hazardous and Solid Waste amendments, 52,231
Hazardous waste
analytical procedures, 263-264
dioxin problems, 1-16
incineration of PCDD and PCDF isomers, 22
Hazardous waste sites
cleanup actions, 8,10-14
United States, 1
Headquarters Permit Assistance Team, 241
Health hazards, photochemical treatment of PCBs, 362-363
Heating element power center, portable infrared incinerator, 316
Heptachlorinated dibenzodioxins, formation, 223t
Herbicide Orange
soil decontamination, 295
See also Agent Orange
Herbicide plant
closing, 224
decontamination, 221-228
recycling production, 222f

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ix002

Herbicide sprayers, cohort studies, 148,150t
Hexachlorinated dibenzodioxins
formation from hexachlorocyclohexane, 223t
See also Polychlorinated dibenzodioxins (PCDDs)
Hexachlorobenzene, human health effects, 71-72
Hexachlorocyclohexane
thermal decomposition, 221
thermal destruction residue, 223t
Hexachlorophene, 74-75,83,259
High-volume sampler, 271f,273
Horses, 2,3,7,8-TCDD analyses in Seveso, 27t
Huber reactor, tests, 15
Human adipose tissue, concentration of PCDD and PCDF isomers, 165t
Human breast milk
dioxin and dibenzofuran levels, 162-172
dioxin toxic equivalents, 168
Human exposure routes, persistent organic chemicals, 99-100
Human health effects
PCDDs, 68
2,3,7,8-TCDD, 7,146t
Human milk, similarity of dioxin and dibenzofuran isomers, 164
Human skin, permeability to xenobiotics, 185
Humans
ingestion of soil, 183t
PCDDs and PCDFs, 27-28
2,3,7,8-TCDD toxicity extrapolation to humans, 54-65
Hydrogen peroxide, required for lignin biodegradation, 341

I

In situ radio frequency heating
cost estimate, 337
decontamination of soil, 332
description, 332
experimental procedure, 333-334
systems, 336
treatment module, 338f
In vitro tests, biological activity of dioxins, 55
Incineration
dioxin wastes, 14-15,42-44
improving efficiency, 15
Incinerator
destruction processes, 9-10
requirements for dioxin, 219
use in Missouri, 219
See also Hazardous waste, Municipal incinerators
Indicator approach, 2,3,7,8-TCDD concentrations, 258
Indicator kriging
description, 249-251
estimator, 252
goal, 251
indicator classes, 251t
use, 252-254
See also Kriging
Industrial site, dioxin contamination problems, 246,248
Ingestion, dioxin-contaminated soil, 181-182
Inhalation, dioxin exposure, 185,189
Inorganic concentrations, advanced electric reactor feedstock, 307t
Insecticide Act of 1910, 231
International Agency for Research on Cancer, 156
International Dioxin Registry, 156

J

Johnston Island
desorption and photolysis tests, 329
permitting costs for small-scale demonstrations, 234t
soil desorption, 323

K

Kriging
description, 247
See also Indicator kriging

L

Laboratory testing, soil decontamination, 293-295
Lake Huron, PCBs, 97-98
Lake Ontario, persistent residues, 96
Lake Superior
model of PCBs, 97t
PCDFs in fish, 26
source of contaminants, 95
Landfills
destruction processes, 9-10
New York, 13
Leaching, tetrachlorinated dibenzodioxins, 6
Lead, amount ingested through dust and dirt, 182-183
Lifetime acceptable daily intake
2,3,7,8-TCDD, 65
See also Acceptable daily intake

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ix002

Lignin
biodegradation, 341
metabolism to carbon dioxide, 344
Ligninases, purpose, 341-344
Localized meteorological regimes, role in air monitoring, 270
Loss-tangent, definition, 335
Love Canal, 2,3,7,8-TCDD contamination, 35

M

Meat, detectable amount of dioxin, 206
Medical studies
current, 157f
nonindustrial, 156t
Mercury arc lamp, detoxification of dioxin, 89
Mercury-excited phosphor lamps, emission spectra, 356f
Military sites, remedial action research, 229
Milk, detectable amount of dioxin, 206
Minnesota Department of Health, 354
Minnesota Pollution Control Agency, PCB contamination, 354-355
Mirex, mineralization, 345t
Missouri
cleanup costs, 12t
dioxin cleanup, 11-12
dioxin contamination problems, 216,259
exposure to 2,3,7,8-TCDD, 74-75
relocation of families, 218
soils, 111
2,3,7,8-TCDD contamination extent, 259
Missouri Department of Natural Resources, 115,217,278,316
Mobility, 2,3,7,8-TCDD, 125
Monitoring wells, 279
Morbidity, Vietnam pilots, 76
Morbidity and Mortality Weekly Report, 175
Mortality data, 2,4,5-T, 72-73
Multistage model, use in dioxin study, 199
Municipal incinerators, 22,24f,48
Mutagenicity studies, Ames test, 196

N

Nasal cancer, association with phenoxy herbicides, 150
National Academy of Science, risk assessment uncertainties, 191
National Cancer Institute, 156
National dioxin study, 34-52
National Environmental Policy Act, 238
National Institute for Occupational Safety and Health, 37,156,217
National Research Council, 96,183
Naval Construction Battalion Center
demonstration test, 304
problems with the advanced electric reactor, 308
soil, 302,320
Neonatal death, children of Vietnam pilots, 76-77
Neurasthenia, 2,3,7,8-TCDD, 71
New Jersey
dioxin cleanup, 13-14
effect of dioxin, 3
Niagara River, 96
No-observable effect level (NOEL), 163,196
Nonparametric geostatistics, 246
Nursing infants, PCDD and PCDF levels, 172

O

Objectives, air monitoring survey, 269
Occupational exposure, retrospective assessment difficulty, 77
Octachlorinated dibenzodioxins
formation from hexachlorocyclohexane, 223t
urban air, 25
See also Polychlorinated dibenzodioxins (PCDDs)
Octanol-water partition, equilibration cell use, 109f
Octanol-water partition coefficient, 2,3,7,8-TCDD, 90,108-110
Office of Solid Waste and Emergency Response, 40
Ohmic heating, mechanism, 335
Ontario, chemical residues, 99t,100
Ontario Ministry of the Environment, 62
Operation Ranch Hand
effect on immune response, 154
See also Ranch Hand Health Study
Organic solvents, dioxin photolysis role, 89
Organohalides, persistence, 341
Oryzias latipes, 134
Oxidation, 2,3,7,8-TCDD, 85-86

P

Parameters, air monitoring survey, 269
Paresthesias, 2,3,7,8-TCDD, 71
Particles, dioxin sorption, 85

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ix002

Particulate matter
  determination, 275
  filter used, 272f
  relationship to dioxin contamination, 275,276f
Paving, remediation measure, 194
Pentachlorinated dibenzofurans
  car exhausts, 24f
  municipal incinerators, 24f
Pentachlorophenol
  effect of dioxin on users, 4
  major contaminant, 21
  mineralization, 345t
  occupational exposure, 69
  source of dioxins, 2
Perimeter sampling, description, 370
Peripheral neuropathy, prevalence among chloracne cases, 154
Persistent toxic chemicals, liability, 102
Personnel monitoring, 305
Personnel protection equipment, advanced electric reactor, 304
Phanerochaete chrysosporium
  biodegradation
    chlorinated organic compounds, 340-348
    lignin, 341
  dechlorination, 346
  degradation of chlorinated compounds, 344
  mineralization of selected compounds, 345t
  2,3,7,8-TCDD degradation, 85
Phenoxy herbicides
  associations with exposure, 158
  relationship to lung cancer, 148
Photochemical reactivity, factors, 358
Photochemical surface decontamination
  PCB application, 350-364
  See also Photolysis
Photochemical treatments, application to PCBs, 363-364
Photochemistry, PCBs, 357-360
Photodegradation, 2,3,7,8-TCDD, 86-90
Photolability, PCDDs and PCDFs, 363
Photolysis
  chlorinated terphenyls, 360
  chloronaphthalenes, 360
  experimental sites, description, 325
  haloaromatic compounds, 360
  laboratory testing and results, 324
  PBBs, 360
  tests
    bench-scale results, 325-326
    generation of scrubber solution, 325
    results, 329
  See also Photochemical surface decontamination
Photolysis rate, effect of organic solvents, 88
Photolysis system, design in thermal desorption-UV photolysis, 327
Photoproducts, PCDDs, 87-88
Photoreduction
  higher chlorinated dioxins, 84-85
  PCBs, 351
Physical constants, 2,3,7,8-TCDD, 105-112
Pica, incidence, 182
Pigs
  soil ingestion, 206
  2,3,7,8-TCDD analyses in Seveso, 27t
Plants
  contamination of aerial parts, 205
  dioxin uptake, 205
  effect on 2,3,7,8-TCDD mobility, 125-128
  sample preparation in mobility study, 119
  2,3,7,8-TCDD accumulation, 128,129t,132-134
Polyarthralgia, 75
Polybromobiphenyls (PBBs), photolysis, 360
Polychlorinated biphenyls (PCBs)
  accelerated photodegradation, 360-362,376
  chemical analysis in photolysis study, 352-354
  chemical degradation methods, 376
  decontamination
    batch process, 378
    continuous and closed process, 382f
    continuous and open process, 380f
    continuous-flow process, 379
    surfaces, 381
  degradation by CDP process, 376-382
  exposure to fluorescent sunlamps, 355
  extinction coefficients, 359t
  field test of photochemical decontamination, 357
  fire incidents, 372t
  irradiation, 352
  Lake Huron, 97-98
  mineralization, 345t
  photochemical surface decontamination, 350-364
  photochemistry, 357-360
  photodecomposition, energy requirements, 355
  photolability, 355
  photoreduction mechanism, 351
  reaction quantum yields, 359t
  sampling on concrete, 352
  stability, 350
  toxicity, 363
  vapor pressure, 97
  See also Aroclor 1254, Aroclor 1260, Aroclor mixtures

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ix002

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ix002

Polychlorinated dibenzodioxins (PCDDs)
accumulation, 5,363
biological activity relative to 2,3,7,8-TCDD, 57t,58t
breast milk levels, 28,166t
characteristics, 6
chemical residues in food, 100
combustion sources, 42-44
crustaceans, 26-27
decontamination
buildings, 367-375
surfaces, 381
degradation by CDP process, 376-382
global distribution, 20-31
hazardous air pollutant, 48
human levels, 27-28,166t
in situ decontamination of surfaces, 382t
isomers, 21t
levels
bovine fat, 29t,166t
breast milk, 28,31t,171
human adipose tissue, 30t
occupational exposure, 69
photolysis, 86-87
photoproducts, 87-88
risk assessment, 5-8
risk-qualified mapping, 246-258
sources, 21,69,101
stack emissions, 45,46t
structures, 20
toxicity equivalents, 9t
See also Dioxin, Hexachlorinated dibenzodioxins, Octachlorinated dibenzodioxins, Tetrachlorinated dibenzodioxins, 2,3,7,8-Tetrachlorodibenzo-p-dioxin
Polychlorinated dibenzofurans (PCDFs)
accumulation in irradiated PCB mixtures, 363
Baltic Sea, 25
chemical residues in food, 100
concentration in human milk, 28,166t
crustaceans, 26-27
decontamination of surfaces, 381
degradation by CDP process, 376-382
global distribution, 20-31
Hudson River, 25
human levels, 27-28
in situ decontamination of surfaces, 382t
isomers, 21t
Lake Superior, 26
levels
bovine fat, 29t
breast milk, 28,31t,171
human adipose tissue, 30t
sources, 21,101
stack emissions, 45,46t
structures, 20
Polyethers, degradation by PCBs and PCDDs, 376-382
Polyethylene glycol, 292
Porphyria cutanea tarda, symptoms, 145
Portable infrared incinerator
advantages, 318
combustion air, 314
construction, 312
data acquisition, 315
exhaust system, 315
feed preparation system, 312
heating element power center, 316
operating conditions at Times Beach, 317t
performance assessment, 311-318
primary chamber, 312-314
process instrumentation, 315t
results of destruction testing, 317
secondary chamber, 314
thermal destruction testing, 316-318
wet gas scrubber, 314
Postreactor treatment, advanced electric reactor, 302
Poultry, 2,3,7,8-TCDD analyses in Seveso, 27t
Primary chamber, portable infrared incinerator, 312-314
Process instrumentation, portable infrared incinerator, 315t
Process parameters, advanced electric reactor, 304t
Proportional mortality ratio studies, description, 147

Q

Quality assurance programs, decontamination, 371
Quality control, ambient air monitoring survey, 273

R

Rabbit
acute toxicity of 2,3,7,8-TCDD, 5t
oral bioavailability of dioxin, 188
2,3,7,8-TCDD analyses in Seveso, 27t
Radio frequency
definition, 335
deposition electrode array, 336
principles, 335-336
Ranch Hand Health Study
description, 158
See also Operation Ranch Hand
Rats
acute toxicity of 2,3,7,8-TCDD, 5t,199
AHH induction, 136,187
dioxin toxicity data, 5t,199
treatment methods in bioavailability study, 135

Reaction quantum yields, PCBs, 359t
Reductive dechlorination
PCBs, 358
See also Dechlorination
Remedial action, dioxin-contaminated sites, 268
Remediation
dioxin-contaminated surface impoundment, 278-285
Missouri sites, 260
program summary, 285
Reproductive outcome studies, 153t
Research development and demonstration
project recommendations, 242
rationale for delisting, 239
permits
comparison of two cases, 238t
difficulties, 241
goals, 237
purpose, 232-233
regulatory problems, 237-238
Residential area, safe dioxin level, 208
Resource Conservation and Recovery Act (RCRA)
dioxin-listing rule, acutely hazardous waste streams, 50-51
hazardous material transport, 311
Huber process, 306
permit, 280
permitting costs
full-scale demonstration, 235t
small-scale demonstration, 234t
requirements
difficulties with dioxin incineration, 233
storage, 218
Risk
considerations, 101
function of two variables, 95
overestimation, 6
PCDD and PCDF isomers, 168
Risk assessment
cancer, 207
dioxin-contaminated soil, 178-201
dynamic nature, 177
EPA model use, 195
examples, 191-194
PCDDs, 5-8
uncertainties, 174-177,191
Risk criteria, adoption, 207
Risk management, dioxins, 9-15
Risk-qualified mapping, PCDD contamination, 246-258
Rosemount Research Center, PCB contamination, 354-355
Rotary-kiln incinerator
dioxin test, 237
use, 260

S

Safety, thermal desorption-UV photolysis, 326
Sample screening techniques, dioxin, 274-276
Sampling
air monitoring survey, 270
buildings, 367-375
design, 368,370
development of a directed program, 369-370
dioxin-contaminated site, 284-285
site selection, 269-270
See also Composite sampling
Scrubber, thermal desorption-UV photolysis, 320-322,326,328-329
Scrubber solution
concentrations after photolysis tests, 329
generation for photolysis tests, 325
Secondary chamber, portable infrared incinerator, 314
Sediments, PCDDs and PCDFs, 23
Semivolatile organic compounds, production from the advanced electric reactor, 308
Sensory neuropathy, 71
Seveso, 3,9,10-11,27t,68,88,96
Sewage sludge incineration, chemical wastes, 101-102
Sheep, soil ingestion, 206
Shirco process, description, 15
Site conditions, advanced electric reactor, 302
Skin depth, definition, 335-336
Sludge
dewatering, coagulation use, 288-289
excavation, 283
treatment, dioxin-contaminated site, 281-282
Smelt, role in ecosystem, 99
Sodium borohydride, 362
Soft tissue sarcomas
Agent Orange, 151-152
relationship to phenoxy herbicides, 150
studies from national census data, 152t
Soil
Agent Orange decontamination, 319-331
analytical methodology comparison for 2,3,7,8-TCDD, 264t
decontamination, 292-295,332-339
deposition on skin, 186t
destroying dioxin-contaminated, 12
detoxification of dioxin with a mercury arc lamp, 89
dielectric properties, 335
differential bioavailability of 2,3,7,8-TCDD, 131-138
dioxin contamination
data, 291-298
German herbicide plant, 225-226
limit, 300

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ix002

Soil--Continued
dioxin exposure from ingestion, 181-185
dioxin-bound, 175-176
hazard assessment, 178-208
ingestion by cows and sheep, 206
mobility
study results, 121-125
2,3,7,8-TCDD, 115
organic compound absorption, 296
partition measurements, 111
PCDDs and PCDFs, 23
photolysis of 2,3,7,8-TCDD, 88
physical-chemical analysis, 323
preparation in mobility study, 116-119
staging pad, 281
2,3,7,8-TCDD contamination, 50,136
thermal desorption, 322,333
thermal desorption-UV photolysis, 320
thermal recovery of tetrachloroethylene, 334-335
treatment methods in bioavailability study, 135
Soil-water partition
regression coefficient, 110t
2,3,7,8-TCDD, 110-112
Soil-water partition coefficients
Missouri soils, 111t
2,3,7,8-TCDD, 112t
Species, 2,3,7,8-TCDD toxicity, 55
Specific activity, dioxin measurement, 106
St. Clair River, contamination, 98
Staging pad, soil, 281
Statistical power, role in epidemiologic studies, 147
Stomach cancer, workers exposed to trichlorophenol, 148
Storage, dioxin contamination, 280,283
Superfund
health assessment manual, uptake of contaminated soil, 184
innovative technology evaluation, 241
national priorities list, 38-40
role in dioxin incineration, 220
See also Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA)
Surface impoundment, remediation measures, 278-285
Surface photoreactor, fabrication in PCB study, 354

T

Tandem mass spectrometry
2,3,7,8-TCDD procedure, 265t
use in thermal destruction testing, 317
Tandem quadrupole mass spectrometer, dioxin analysis, 262
Task force on risk assessment and risk management, 176
TCDD--See 2,3,7,8-Tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD)
Technical products, source of PCDDs and PCDFs, 21
Tetrachlorinated dibenzodioxins
Missouri, 11
photolysis rates, 87
toxicity, 5
See also Polychlorinated dibenzodioxins (PCDDs)
2,3,7,8-Tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD)
abiotic transformation, 86-90
absorption spectra, 86
acceptable daily intake, 163
action level in soil, 179
acute effects, 70
AHH induction marker for exposure, 138
air monitoring, 219
analysis, 260
analytical determinations, 264t
analytical support at contaminated sites, 259-265
application of nonparametric geostatistics, 254
background sites, 49
bioavailability, 133,136
bioconcentration in plant matter, 129t
biological transformations, 85-86
cancer risk estimates, 197t
cleanup, 41
concentration
as a function of depth, 126t
indicator approach, 258
on particulate matter, 275
concentration profile
control side, 123f
damp conditions, 122f
saturated conditions, 122f
shaded side, 123f
unshaded side, 124f
contamination, 35,74-76
content of plant matter, 127t
cow metabolism, 207
degradation, 380f
differential bioavailability from contaminated soils, 131-138
effect of distribution at the site, 189
effect of treatment conditions on thermal desorption tests, 328t
effect on liver, 71
efficiency of portable infrared incinerators, 318t
elimination from decane solution, 377

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ix002

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ix002

2,3,7,8-TCDD--Continued
environmental chemistry, 82-91
equivalents
adipose tissue, 169
chlorinated dibenzofurans, 167
definition, 164-168
human milk, 169t
potency factors used, 38t
extrapolation to humans from chronic animal studies, 64t
formation, 131,223t
hazard of synthesis, 71
human health effects, 146t
industrial exposures, 146
industrial site contamination, 193-194
levels in plants, 125-128
measurement of vapor-phase photolysis, 89
microbial metabolism, 85
Missouri, contaminated soil, 216
mobility, 114-129,248
neurasthenia, 71
no-observed adverse effect level, 62
octanol-water partition coefficient, 90,108-110
oxidation, 85-86
paresthesias, 71
pesticide manufacturing facilities, 49
photodegradation, 86-90
photolysis
on glass surfaces, 88
reaction rates, 325
physical constants, 105-112
plant uptake, 128
poisoning outbreaks in the general population, 74-77
probabilities and estimates, 257t
problems, 143
prohibition of residue transport, 224
properties, 83
pyrolysis, 302
remedial action research at military sites, 229-242
removal from waste water, 286-290
Saginaw Bay, 25
soil movement, 90-91
sources, 47t,83-85,248
stability, 91
symptoms of exposure, 68
synthesis, 82
toxicity, 36-37,54-65,267,134
toxicity study in rats, 63t
transport processes, 90-91
treatment conditions during thermal desorption tests, 324
tumor
initiation, 195-196
production mechanism, 65
promotion, 62,163

2,3,7,8-TCDD--Continued
uptake by route of exposure, 181t
UV degradation, 377
vapor pressure, 91
volatility, 114,125
water solubility, 105-107
See also Dioxin, Polychlorinated dibenzodioxins (PCDDs)
Tetrachloroethylene
recovery from soil, 334
thermal recovery from soil, 334-335t
Thermal decontamination, soil, 333
Thermal desorption
description, 15
effect of treatment conditions on soil, 324t
laboratory testing and results, 322-324
soil decontamination effectiveness, 331
tests at Naval Construction Battalion Center, 327
Thermal desorption-UV photolysis
effect of soil temperatures, 327-328
on-site pilot testing, 326
photolysis reaction kinetics, 330f
process
concept, 321f
description, 320-322
soil decontamination, 319-331
Thermal destruction testing, portable infrared incinerator, 316-318
Times Beach
bioavailability of 2,3,7,8-TCDD, 134
dioxin research station, experimental design, 115
dioxin thermal destruction testing, 316-318
effect of dioxin, 3
evacuation, 217
mobility of 2,3,7,8-TCDD, 114-129
Titanium dioxide, 362
Toxic chemicals, persistence, 94
Toxic organic waste, destruction, 94-103
Toxic Substances Control Act (TSCA)
description, 231
Huber process, 306
small-scale demonstrations, 231,232t
Toxicity
animal data extrapolation, 7
decontamination reagents, 292t
dioxins, 54
2,3,7,8-TCDD to humans, 5,54-65
See also Extraction procedure toxicity
Toxicity equivalent factor, 8,36-37,59,60t
Toxicity testing, aromatic halides, 292
Transport processes, 2,3,7,8-TCDD, 90-91

1,2,4-Trichlorobenzene, thermal decomposition, 221
2,4,5-Trichlorophenol (2,4,5-T)
  contamination, 48
  destruction ability, 306
  formulated pesticidal products, 42
  formulators, 41-42
  major contaminant, 21
  morbidity studies, 72
  mortality data, 72-73
  production, 3,70,221
  risk of soft tissue sarcomas, 73
  source of dioxins, 2
  teratogenicity, 230
2,3,5-Trichlorophenoxyacetic acid, 83,131,248
Triethylamine, use in photocyclization, 362
Trifluoroacetic acid, 362
Trifluralin, absorption coefficients, 353t
Triplate transmission line, use, 336-337
Tumor, promotion versus initiation, 194-195

U

U.S. Air Force, 231,319
UV light
  PCB absorption, 357-358
  PCB reactivity, 351

V

Vapor pressure
  PCBs, 97
  2,3,7,8-TCDD, 91
Variogram, description, 247,252
Vertac Rule, 51
Veterans Administration, 156
Vietnam
  Agent Orange use, 300
  analysis of soil samples, 23
  breast milk levels of dioxin, 168
  PCDD and PCDF levels, 28
  pilots, morbidity, 76
  veterans, excess birth defects, 153
Virtually safe dose
  Canada versus Europe, 196
  dioxin, 163,195,205
Volatilization, 2,3,7,8-TCDD, 90,114

W

Walkely-Black method, 110
Waste cleanup, dioxin as a model, 2
Waste treatment systems, 346-348
Waste water
  2,3,7,8-TCDD removal, 286-290
  See also Well water
Water solubility
  radiolabeled dioxin, 105-106
  2,3,7,8-TCDD, 105-107
Water treatment systems, dioxin contamination, 280
Well water
  2,3,7,8-TCDD removal, 286-290
  See also Waste water
Wet gas scrubber, portable infrared incinerator, 314

X

Xenobiotics, initial oxidation, 344-346

Production by Paula M. Bérard
Indexing by Keith B. Belton
Jacket design by Carla L. Clemens

Elements typeset by Hot Type Ltd., Washington, DC
Printed and bound by Maple Press Co., York, PA

Publication Date: April 24, 1987 | doi: 10.1021/bk-1987-0338.ix002